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THE FRONTAL SINUS: OPENING IT THROUGH THE NOSE FOR CHRONIC SUPPURATION.

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As late as 1905 the intranasal opening of the frontal sinus was thought too dangerous to be attempted. Since then, however, E. F. Ingals, Max Halle, Vacher, H. P. Mosher and lately P. Watson Williams and Herbert Tilley have proved that the frontal sinus may be opened for drainage through the nose with no greater risk than that of the external frontal sinus operation.

The following article combines present views with original observations. To Professor Basil Harvey, of the department of anatomy, of the University of Chicago, I am indebted for the opportunity he has granted me for the anatomical study of the subject.

Anatomy. The surgical anatomy of the frontal sinus necessarily includes that of the anterior ethmoid region. Both the sinus and its ethmoid neighborhood present many well known abnormalities such as absence of one or both frontal sinuses, minuteness or excessive size of a sinus, coalescense of both sinuses into one, or of the ethmoid cells into large simple cavities and the existence of a large frontal bulla (Fig. 1, Fig. 5). These abnormalities would make the operation hazardous but for the information supplied by the X-ray and the constancy in structure of the main walls of the surgical approach to the sinus from the

nasal cavity, which makes variations in the arrangement of the anterior ethmoidal cells included by these walls of less importance. In addition, Hajek's fundamental lamellae, the uncinate process, the bulla lamella and the middle turbinate, are always present to guide the operator, unless removed by disease or previous operation.

Relations of the Frontal Sinus Ostium. In most examples of the detached frontal bone the openings of the frontal sinuses are seen to be quite large enough for drainage (Fig. 2). The apposition of the ethmoid cells to the frontal bone, however, besides imposing upon the sinus a narrow and easily blocked ethmoidal

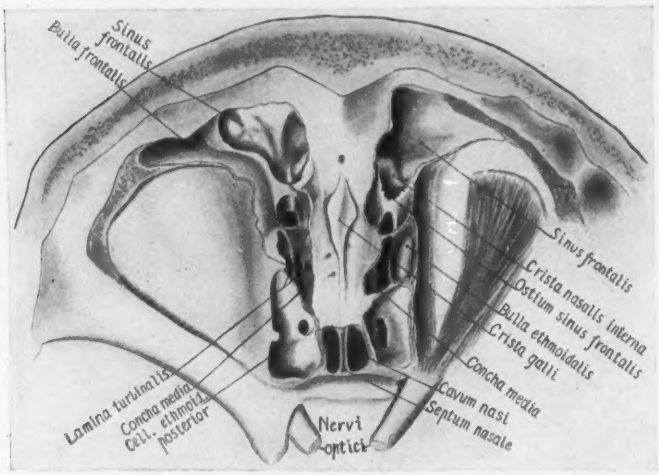

Figure 1. The floor of the anterior cranial fossa has been removed showing the orbits, frontal sinuses and ethmoidal tracts opened from above. The peri-orbita covers the ocular bulb in the left orbit; the bulb is exposed in the right orbit.

outlet, the fronto-nasal duct, (Fig. 3, Plate B), reduces the naturally wide frontal sinus opening in the frontal bone, (Fig. 4B), to the typically small sinus ostium, (Fig. 4A), or upper entrance into the fronto-nasal duct. The ostium lies at a lower level than the frontal bone opening of the sinus, at the bottom of a funnel formed by the walls of the anterior ethmoid cells and by the internal angular process (internal nasal crest), (Fig. 1, Fig. 3), of the frontal bone, which converge to form the floor of the sinus about the ostium. Normally the ostium is bounded in front by

the internal angular process of the frontal bone. Exceptionally an anterior ethmoid cell lies between the ostium and the nasal crest, displacing the ostium farther back. Externally and posteriorly an ethmoid cell or cells lie between the ostium and the orbit.

Backward from the ostium extends the chain of anterior and posterior ethmoid cells, (Figs. 1, 2, 3, 5, 6), which, beginning in front with the bulla ethmoidalis, or a cell in front of it, ends behind at the sphenoid sinus. The uppermost of these cells are incomplete in the ethmoid bone and are completed in the articulated skull when roofed by the innermost portion of the orbital plate (orbital process) of the frontal bone, (Fig. 6c,c), which borders the ethmoid notch (incisura ethmoidalis, Fig. 2), and which is marked by depressions or foveolae, (Fig. 2), which fit over the open ethmoid cells. For this reason I refer to these cells as foveal cells.

Internally the ostium is bounded by the expanded plate of the anterior superior nasal spine of the frontal bone, (Fig. 2), and the turbinate wall of the ethmoid bone. At the posterior part of the inner wall of the ostium funnel is situated the thin part of the orbital process of the frontal bone, (Fig. 6, f), which forms the anterior boundary of the olfactory fossa, whose floor is the cribriform plate, (Fig. 6, e). Where the cribriform plate extends well forward it carries this thin part of the orbital plate forward, also, so that it projects into the frontal sinus as a columnar prominence which is called the crista olfactoria of the frontal sinus by Boenninghaus. Where this prominence is very marked it is called a torus olfactorius by L. Onodi.

The torus region is dangerous surgically because a perforation or fracture, opening into the olfactory fossa just above the cribriform plate, might be made from the sinus through the thin bone of the torus; and for this reason, in enlarging the frontal sinus ostium for drainage no cutting should be done in an inward direction toward the septum interfrontale; moreover, the space gained in this direction would be small.

Enlargement of the ostium anteriorly, because directed away from the brain and orbit, has been favored by rhinologists in spite of the fact that the strong internal nasal crest bars the way in this direction. However, cutting forward is only apparently safer than cutting backward for the hard bone of the nasal crest requires strong instruments for its cutting whose forcible manipulation endangers the often overhanging posterior sinus wall. The most used of these instruments is the rasp, which, to cut effectively

downward, must necessarily be pushed or even wedged strongly up into the sinus. I have been informed of one death from meningitis following the use of the rasp.

The space gained in enlarging the ostium forward will depend upon the prominence of the internal nasal crest removed and the possible, but rare, helpful presence of an ethmoidal cell following out the crest in front of the ostium. Usually the space obtained is

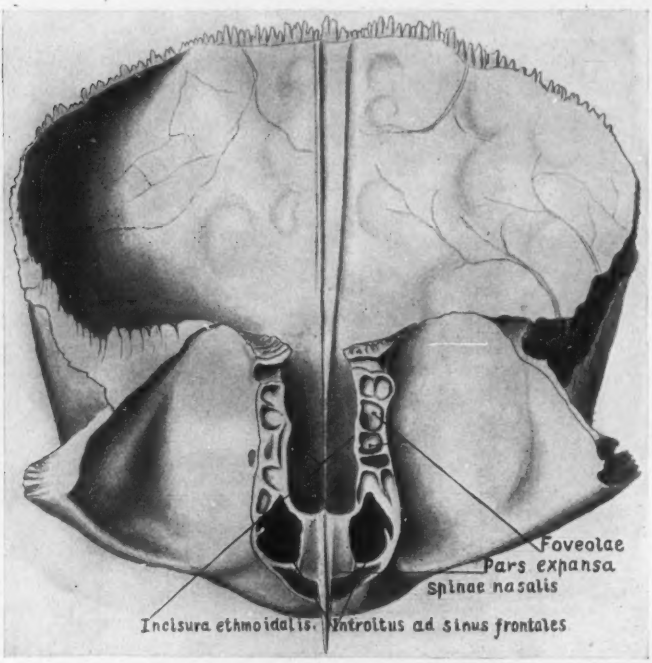

Figure 2. (After Gray.) The frontal bone, showing its orbital surface.

not of itself enough for drainage and later may become lost because of the formation of granulations and cicatricial bands, which replace the destroyed mucous membrane covering of the denuded nasal crest. While the possible enlargement of the ostium forward is, as shown, usually moderate a great increase in its size is obtainable backward by removing the foveal cells mentioned from underneath the orbital plate of the frontal bone forming their roof; no powerful instruments are needed, but merely light

curettes of adapted form, (Fig. 7 a, b, c.). This procedure removes the anatomical obstacle to drainage, the anterior ethmoidal cells and opens the frontal sinus ostium to the full capacity of the frontal sinus opening in the detached frontal bone. The enlargement is made, not in the foremost, narrowest part of the naris, as in cutting forward, but in a capacious part of it. Another advantage is that, as P. Watson Williams says, remnants of mucous membrane in the opened ethmoid cells act as islands from which the reproduction of the mucosa may proceed, giving the broad, new channel into the sinus a mucous membrane lining with no tendency to cicatricial contraction. The enlargement in the posterior direction by breaking down the ethmoid cells is also the way naturally adopted as most effective when the sinus is opened into the nasal cavity from above in the external operation, the surgeon unhesitatingly choosing this direction as the best and not fearing to take it because in the external operation he can easily see where he is going. Unless thoroughly familiar with the anatomy of the region those attempting the intranasal operation shun the chief obstruction to the sinus, the anterior ethmoid cells and attack the nasal crest only, so getting an opening that is too small for drainage and closes readily by granulation.

The Operative Field. Looking from above into the anterior cranial fossa the cribriform plate is seen to form the floor of the more or less deep olfactory fossa, (Fig. 6, e), which is divided sagittally by the crista galli into two parallel oblong excavations, shallow behind but growing narrow and deep toward their front, where they end at the posterior wall of the frontal sinus, and, as described, may help to form the inner wall of its ostium. The walls of the olfactory fossa are formed from the innermost part of the orbital plate of the frontal bone. They rise gently from the edges of the cribriform plate behind and more and more steeply towards its front, so that they may actually overhang the olfactory fossa along its anterior half. After thus ascending from the cribriform plate in walling in the olfactory fossa the orbital plate bends over to a horizontal level on each side of the fossa to cover the foveal row of ethmoid cells mentioned, rising thence rather steeply to form the irregular vaults that roof the orbits. The portion of the orbital plate covering the foveal cells is usually about three-eighths of an inch wide, (Fig. 6, c, c,). A posterior extension of the frontal sinus in the form of a recessus paracribrosus of L. Onodi, may displace the foremost foveal cells backward.

In very many cases the foveolae of the foremost one or two ethmoid cells, instead of remaining mere depressions along the border of the ethmoid notch of the frontal bone, become cavities penetrating the orbital plate of the frontal bone in the form of fronto-ethmoidal cells which arch outward, more or less, over the orbit, (Fig. 1). The foremost cell may thus extend outward as a frontal bulla even beyond the frontal sinus and greatly exceed its capacity, forming what looks like a second frontal sinus behind the usual one, (Fig. 1). In these not uncommon cases the orbital process of the frontal bone is divided horizontally by the fronto-ethmoidal cells into two lamellae, one arching over the eye and

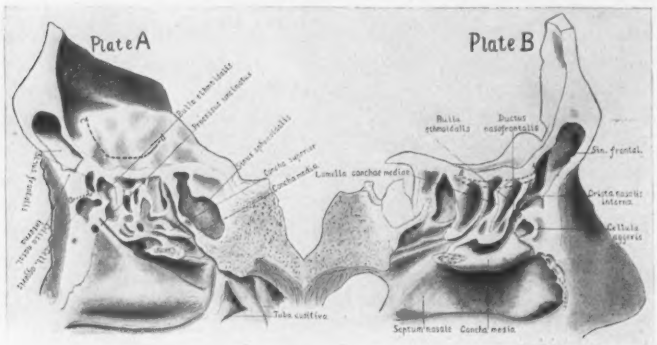

Figure 3. A sagittal saw-cut through the ethmoid cells, showing the surgical passage of approach laid open. In plate A is seen its outer wall with the lamina papyracea forming. the external limit of the ethmoid cells displayed; in plate B is seen its inner wall, the turbinal plate, forming the internal limit of the same cells and ending below in the middle turbinate whose outer surface is shown together with the fronto-nasal duct leading up into the frontal sinus. a, b, level of ostium of frontal sinus; c, d, line of demarcation between the ethmoid cells and orbit; e, f, level of cribriform plate.

the other over the large fronto-ethmoidal air cells. Excepting intranasal drainage such cells are beyond surgical interference if suppurating.

The portion of the orbital process of the frontal bone covering the anterior ethmoid cells is continuous with the posterior cranial wall of the frontal sinus with which, seen in profile, (Fig. 3), it forms a sweeping curve, and in clearing away the obstructing ethmoid cells from the sinus interior into the nose, the curette follows this curve. Although thin, this cranial covering of the ethmoid cells is of hard, firm bone, and, unless directly attacked

in operating, it is in no danger of perforation. A curette of the form shown in Fig. 7, a, b, will sweep away cell partitions backward from underneath it, gliding over it without penetration. From Figs. 3 and 5, it is evident that where the cell partitions are cleared away under the orbital plate a wide space is established from the middle meatus of the nose up into the frontal sinus.

The outer wall of the operative field is the lachrymal bone in front and the lamina papyracea of the ethmoid bone behind. This, the orbital wall of the field, is often frail and is liable to perforation if instruments are directed too far outward and used with pressure. I have heard of two perforations of the orbit occurring in operations upon the ethmoid cells, one resulting in orbital abscess and the other in an intra-orbital arterial hemorrhage with immediate exophthalmos. Blindness of the eye involved resulted in both cases. In a third case a perforation in the lachrymal region had no bad results.

Inwardly, the operative field is bounded by the turbinal wall, (Figs. 1, 3, 5), of the ethmoid labyrinth which is continuous above with the lateral wall of the olfactory fossa in the cranial cavity, so that a fissure created in the upper part of the turbinal wall might lead to meningitis. The turbinal wall terminates in the middle turbinate below. P. Watson Williams speaks of the turbinal wall simply as the "vertical plate." This is confusing, as the perpendicular plate of the ethmoid bone is also spoken of as the vertical plate and it is better to use the distinctive term, turbinal wall, employed by Hajek, as this wall gives rise to both the superior and middle turbinates.

The Intranasal Approach to the Frontal Sinus. The often described narrow and often tortuous anatomical way from the nasal cavity to the sinus, the nasofrontal duct and infundibulum, (Fig. 3), ascends underneath the middle turbinated body among anterior ethmoid cells to the sinus ostium. The broader surgical passage of approach lies between the parallel walls mentioned, the orbital wall and the turbinal plate of the ethmoid bone and disregards the ethmoid cells in the way. The space between these walls, which I call the surgical passage of approach, extends antero-posteriorly from the ascending frontal process of the superior maxillary bone in front, to the lamella of the middle turbinate in the ethmoid labyrinth behind, (Fig. 3), this lamella separating the operative field from the posterior ethmoidal cells. In its narrowest part the passage of approach is from one-fourth to one-half of an inch wide.

P. Watson Williams says: "The width of this potential passage between the outer and inner boundaries at its narrowest part

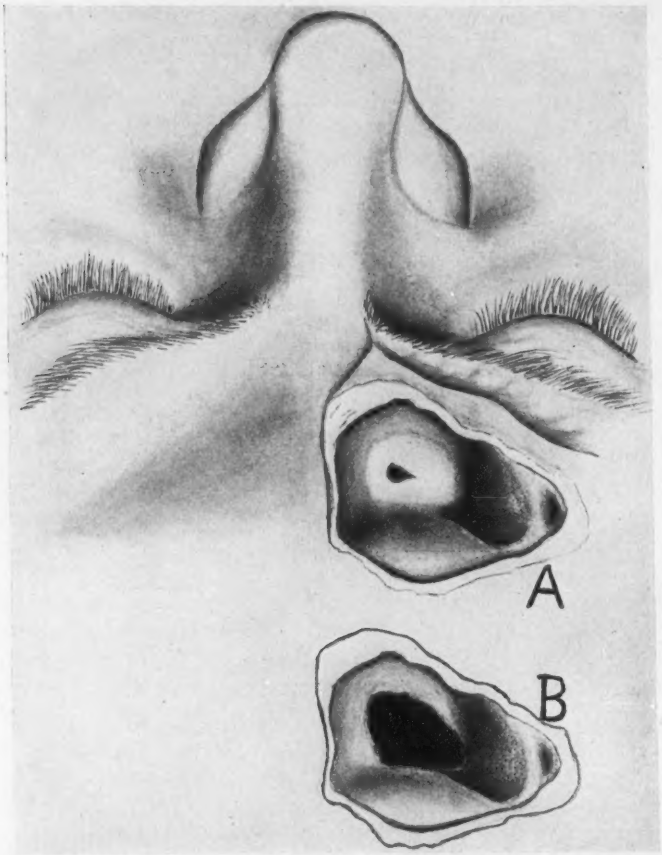

Figure 4. Part of face of a cadaver seen from above and behind the head, the observer looking down into the opened frontal sinus showing A, the ethmoidal cell part of the sinus floor intact, with the ostium; B, the same sinus after the ethmoid cell part of its floor has been removed from above with the author's frontal sinus curettes.

measures in the adult seven to twelve millimeters. The width of instrument needed to clear the space should not exceed six millimeters."

The lower limit of the passage of approach is indicated by a line drawn through the bottom of the bulla ethmoidalis and just below the agger nasi, the floor of the bulla forming the lowest part of the ethmoid labyrinth, (Fig. 3).

The roof of the passage of approach to the frontal sinus, the orbital plate of the frontal bone, has already been described.

The operative field, situated in the highest, narrowest part of the nose, (Fig. 5), is often made still more inaccessible by a horizontal deflection outward of the nasal septum involving the lower border of the perpendicular plate of the ethmoid bone, which, with the cartilage of the septum creates an angle that hides even the middle turbinate from view. Such deflections must be resected as a preliminary to the operation.

THE OPERATION.

Probing the Frontal Sinus. A probe should be introduced as a guide into the sinus, either by the natural passage, or, if this be impassible, after clearing away obstructing ethmoid cells. H. P. Mosher showed, in 1912, that almost any frontal sinus may be entered with a probe if the anterior ethmoid cells are previously removed.

In attempting to pass the probe by the natural passage it may find the lower ostium of the fronto-nasal duct in the infundibulum, "ostium infundibulare" of Bönninghaus, or the ostium may lie in front of the infundibulum, opening into the recess extending upward under the middle turbinate towards the frontal sinus and called the recessus frontalis, by Killian. In this case the ostium is called "ostium recessuale."

Hajek says that the chief obstacle to probing the frontal sinus with the middle turbinate in place is the deflection of the probe's direction outward towards the orbit by the lower border of this turbinate, so that a stiff probe, pushed hard in this direction might penetrate the orbit. For this reason, he says that a previous resection of the anterior part of the middle turbinate is usually needed to make probing possible and that even then the upper border of the inferior turbinate tends to direct the probe too far outward. This outward direction may also lead the probe into an infundibular cell instead of the sinus.

The frontal sinus probes in common use are faulty. A good probe should thicken evenly as does a trout rod, from its distal end towards its butt, so that its stem will have enough elastic firmness to control its flexible tip. Copper is the best metal. Silver is too soft. I have designed three probes, a fairly limber one of

copper, a stiffer one of soft untempered steel and a probe with a stiff steel stem which ends in a flexible copper tip, (Fig. 7 e, f).

The operation proper is begun variously by different operators. Mosher formerly broke into an anticonchal, infundibular cell, which he calls the agger cell, (Fig. 3; Fig. 5), and which lies under the

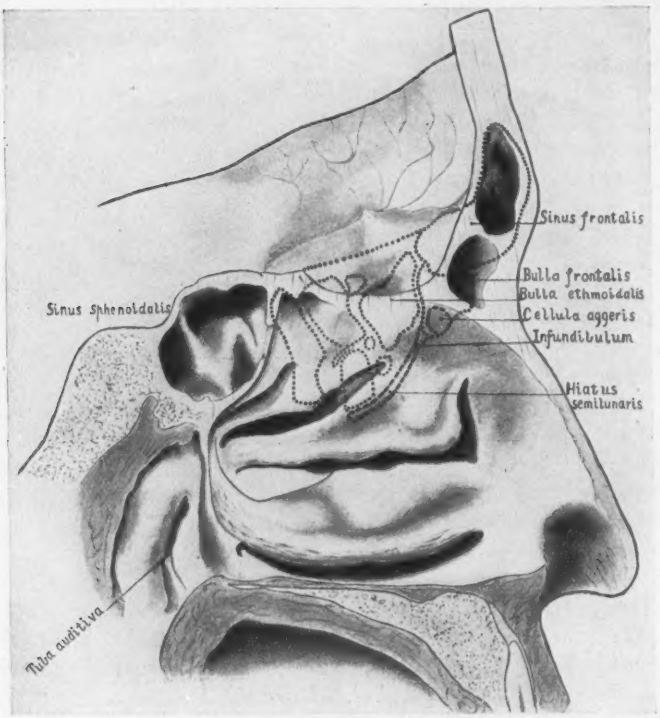

Figure 5. Lateral view of a nasal fossa. The location of the ethmoid cells, frontal sinus and infundibulum is indicated by dotted lines. The exact position of these structures in relation to the lateral wall of the nasal fossa was ascertained by drilling from a sagittal saw cut toward the nasal interior.

agger nasi, or as it is put by Herbert Tilley, in the anterior, superior third of the uncinate process. Mosher found that in most cases he could pass from this cell, when opened, up into the frontal sinus, that is, as I have found it, destruction of this cell breaks into the fronto-nasal duct from in front and below, (Fig. 3), By opening the agger cell, Mosher severed the attachment of the middle

turbinate making it movable so that he did not resect it but left it entire.

The penetration of the ethmoid labyrinth through the agger cell involves cutting through the turbinal plate high up, so near the cribriform plate that a possible fissuring fracture created by the curette used might penetrate the cranial cavity, as is evident from the illustrations showing the agger cell in this article, (Fig. 3, Fig. 5). Mosher has abandoned the agger cell as an entrance to the labyrinth as too inconstant a structure and penetrates the ethmoid cells posterior to it. He still cuts through the turbinal plate, in its upper part. In this regard P. Watson Williams says: "With an infective purulent discharge we should avoid opening up a bare tract so close to the cribriform plate and I always keep intact the upper part of the vertical (turbinal) plate, which is the first part that Mosher destroys."

In addition it is to be considered that the branches of the olfactory nerve lie in grooves in the inner surface of the turbinal plate and that each filament is accompanied by a prolongation of the dura mater and pia mater, so that injury to the upper part of the turbinal plate involves the danger of direct communication of a resulting inflammation with the envelopes of the brain.

P. Watson Williams begins his operation by cutting with forceps through the anterior margin of the middle turbinate where it is attached to the outer nasal wall, stating that the same cut also opens the anterior ethmoid cells, in front of the fronto-nasal passage. It has not been my experience that this entrance may be relied upon. Herbert Tilley removes the anterior part of the middle turbinate. Bönninghaus resects the anterior part of the middle turbinate including the pars fixa, that is the part that covers the recessus frontalis. He says that the middle turbinate resection and uncinate process resection, which he performs often open a cell and show the way into the sinus. Max Halle cuts through the anterior attachment of the middle turbinate, but preserves the turbinate entire.

I have found that in a wide naris, not infrequently, the middle turbinate flanges out so much towards the septum that the whole middle meatus, with the bulla and uncinate process lie in plain view and that in such cases not only probing, but the entire operation may usually be done without cutting even the anterior attachment of the middle turbinate.

Where the olfactory fissure is wide, and the middle turbinate is closely applied to the outer nasal wall, cutting through the anterior attachment of the turbinate, as is done by Mosher, Williams and

Halle, will permit a broad exposure of the infundibular region by pushing the turbinate over against the septum, but in a high narrow naris, where the middle turbinate fits in snugly between the septum and the infundibular region, the loosened turbinate can not move towards the septum and in such cases it is necessary to cut away the anterior half of the turbinate to expose the infundibulum. It is thus evident that, according to conditions, it may be best to leave the middle turbinate intact, cut through its anterior attachment or resect it more or less extensively.

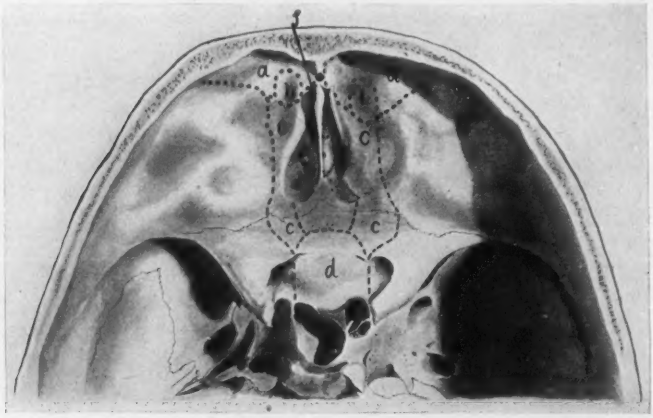

Figure 6. View of anterior cranial fossa showing by dotted lines a, a, the extent of the frontal sinuses; b, b, the location of their ostia; c, c, the rows of foveal ethmoid cells on either side of the olfactory fossa; d, the sphenoidal sinuses (opened from above in the sella turcica); e, cribriform plate; f, the thin-walled foremost part of the olfactory fossa which projects into the frontal sinus in the form of a crista olfactoria or of a torus olfactorius.

If the probe could not be passed its introduction should be again attempted after the middle turbinate has been freed or partly removed. Whether it will then pass or not the next step of the operation, the removal of the anterior ethmoid cells up to the sinus floor, is begun by breaking into the bulla ethmoidalis. The bulla, (Fig. 3), being formed from one of the fundamental ethmoid lamellae (Hajek) is, unlike the cells anterior to it, a constant structure. Whether single or partitioned, to quote Bönninghaus, "it reaches high up to the roof of the ethmoid cells and offers the surest way to the sinus frontalis." The bulla is a large, usually prominent cell easy to find just behind the uncinate process, where it lies in the

most accessible part of the middle meatus. It is usually easily broken into with the curette and it has this great advantage as a starting point, that, because it forms the lowest part of the ethmoid labyrinth, entrance into it may be effected through its bottom which permits directing the curette obliquely upward and outward instead of directly outward towards the orbit, as in the case of the agger and its associated infundibular cells, which must be entered from the side and whose often thick walls resist the curette, making it enter with plunging force, aimed at the orbit. Before entering the bulla it may be necessary to expose it better by resecting the processus uncinatus by means of my septum knives. Bönninghaus resects it as a part of his regular procedure.

The bulla is entered with the cutting edge of the curette turned forward, the curette then sweeping away the anterior ethmoidal cells clear to the ascending process of the frontal sinus. This forward direction of the curette is an advantage over cutting backward from a foremost cell like the agger cell, for the direction forward is a safe one which permits vigorous work, as it aims at the strong bone of the ascending process and internal nasal crest instead of the fragile posterior ethmoid cells with their intimate relation to the optic nerve, (Fig. 1), as is the case in cutting backward. The complete removal of the anterior cells from the bulla forward gives the maximum passage obtainable between the orbital walls limiting the operative field while the important turbinal wall is left intact. The operator must maintain a strictly postero-anterior direction, using as guides the inner canthus of the eye and the ascending process of the superior maxillary bone. An outline of the frontal sinuses drawn in ink upon the patient's forehead from the X-ray plate, is also useful. The clearing away of the anterior ethmoid cells at once admits the probe into the sinus even if it would not pass before. Finally, remnants of cells are cut away with the Grünwald punch.

In extensive hyperplastic ethmoiditis all of the usual landmarks, the bulla, the uncinate process, the hiatus semilunaris are absent, the ethmoid labyrinth being replaced by a formless mass of polypi and fragments of cell walls. Chronic frontal sinus suppuration is often associated with this polypoid destruction of the ethmoid cells which is, however, a help rather than a hindrance to the surgeon for the diseased labyrinth is easily removed with the punch and curette and if the curettage be carried well forward and upward, a broad way is opened into the frontal sinus with surprising ease by simply extirpating all polypoid tissue encountered.

The Enlargement of the Sinus Ostium. In most cases the removal of the anterior ethmoid cells, as described, at the same time

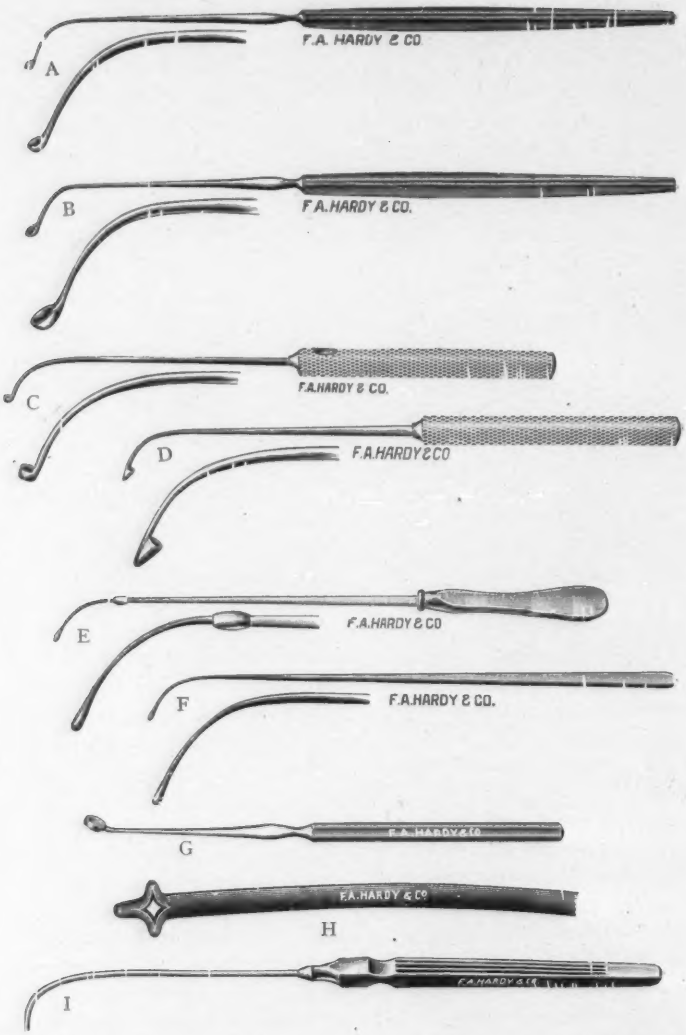

Figure 7. (a) Freer back-cutting frontal sinus probe curette; (b) larger size of the same curette. (c) back-cutting ring frontal sinus curette; (d) forward-cutting frontal sinus curette; (e) frontal sinus probe with steel shank and copper end; (f) copper frontal sinus probe; (g) Freer ethmoid ring curette; (h) Freer intranasal self-retaining frontal sinus rubber drain tube; (i) introducer for drain tube.

breaks away the posterior ethmoid cell part of the frontal sinus floor, (Fig. 4, b), so establishing at once a good opening into the sinus and permitting the entrance into its cavity of burrs or curettes as desired. Not seldom, however, the sinus floor is not only hard and firm in the region of the internal nasal crest but, also, because of the thickness of cell walls taking part in its formation, it forms a hard plate all around the ostium, (Fig. 4, a), so that, while the ostium will admit so small a thing as a probe, the curette used in removing the anterior ethmoid cells, being too large to pass through the ostium and unable to break through the firm sinus floor from below, strikes vainly against it from underneath. For such cases I have devised a probe curette, (Fig. 7, a), a minute sharp spoon which, passing like a probe through the ostium into the cavity of the sinus, when withdrawn, cuts its way out downward and backward, making room for the introduction of a larger, also down and back-cutting, curette of the same form, (Fig. 7, b). This cuts away all of the ethmoidal cell part of the sinus floor and removes remaining cell walls from under the orbital plate of the frontal bone, so establishing a large opening into the sinus which follows the posterior sinus wall and cranial roof of the ethmoid cells in a single sweeping curve, (Fig. 3). These curettes I have designed after much experiment and improving change of form. They glide with perfect adaptation downward and backward under the cranial roof of the anterior ethmoid cells, merely attacking what projects from it without endangering it. They cut in a safe direction downward and backward out of the interior of the sinus into the nasal cavity, as in the external operation. To work upward from below into a sinus of this kind with a hard floor by means of a rasp, curette or burr is not so safe, as it is possible to go astray, and also to endanger an overhanging posterior sinus wall by plunging of the instrument into the sinus through the sudden yielding of the resistence of its floor.

In cutting out from the sinus the curette must not be directed inward towards the septum interfrontale for the reasons given.

In addition to the back-cutting curettes I have designed one which has its edge directed downward and forward to cut away a possible cell lying between the ostium and internal nasal crest, (Fig. 7, b).

When the cutting downward from the sinus with the curettes is finished a communication will have been established between the sinus and nasal cavity, (Fig. 4), much larger than could be gained by cutting forward only with rasp or burr. In this connection P. Watson Williams says: "In the majority of cases I have not found

it necessary to reduce the nasal crest with rasp or burr, the simple removal of the fronto-ethmoidal cells and in some cases biting off of a thin projecting anterior margin of the ostium affording easy passage for a six millimeter bougie."

Nevertheless, cutting away the internal nasal crest with the burr may prove indispensible in the occasional cases where an acute angled crista nasalis interna projects so far backward that even after removal of anterior ethmoidal cells the opening established is insufficient. In such a case a straight burr, such as one of Halle's cylindrical ones should be used to enlarge the sinus ostium forward. To introduce the burr, the place where the stem of a probe in the sinus is lost to view in the tissues in looking upward and backward under the dorsum nasi, should be fixed in view, as this is the location of the internasal crest. The burr is introduced here to a slightly higher level than the crest and pressed forward against it before turning on the power. This precludes injury to the posterior sinus wall. The burr should not be pushed upward while it is cutting, lest it cut into the posterior wall of the sinus. A guard is not necessary; there is little room for it and it complicates the act as most such safeguards do. Halle no longer uses a guard for his new operation but has the ends of his burrs polished. The burr should fit in a dental handpiece, not a heavy surgical one which hinders fine work.

A special set of bougies, to determine the size of the opening made, does not seem to me necessary. The amount of free motion of the stem of the curette or probe in the opening made will indicate sufficiently whether it be large enough.

Suppurating fronto-ethmoidal cells which arch over the orbit will usually be opened freely for drainage by the intranasal operation together with the frontal sinus.

I do not approve of curetting away the mucous lining of the frontal sinus as a last step of the operation, although it is advised by Herbert Tilley and Halle. Such destruction of the sinus lining is based upon the theory, fostered by most textbooks, that the chronic suppuration has led to hopeless degeneration of the mucous membrane. Experience has shown me that the pathologic changes described as always present, such as excessive hypertrophy, polypoid degeneration, fungoid granulations are rare, and that even if these changes do exist they are capable of disappearance under the influence of good drainage and ventilation, unless there be caries of the bone or large polypi present. To systematically destroy the mucous lining by curettage is to replace the mucosa with its

unreproducible epithelial lining by granulation tissue and so unduly prolong the period of suppuration. It is also to be remembered that, unlike the external and especially the Killian operation, obliteration of the frontal sinus is not to be hoped for from the intranasal operation which, besides, gives no access for curettage of the deep recesses of a large frontal sinus. If, in spite of intranasal drainage, the suppuration and headache continue, indicating irremediable pathologic changes in the sinus, the external operation should be resorted to with its advantage of through drainage and good inspection. Failure of the intranasal operation may also result from poor drainage due to more or less complete septa dividing the frontal sinus and because caries exists, usually affecting the sinus floor over the orbit and causing lid swelling and sometimes exophthalmos.

In conclusion, Max Halle's now completed method of opening the frontal sinus intranasally should be mentioned. Halle makes the remarkable claim that his last operation opens the sinus so freely that every bay and niche of its interior may be inspected as easily or more so than in the external operation. The chief features of Halle's new operation are a mucosa-periosteum flap made over the inner surface of the ascending process of the superior maxillary bone and reflected downward and backward to expose this bony process. The anterior attachment of the middle turbinate, the agger nasi and a part of the ascending process of the superior maxillary bone are then chiseled away. Halle claims for this procedure a free access to the ethmoid cells and to the sinus above them unobtainable in any other way.

P. Watson Williams sees no advantage in Halle's flap which he finds "worse than useless," except in cases where it is necessary to shave down an inward projecting edge of the superior maxilla. I have performed Halle's flap operation upon the cadaver, but did not gain enough space to compensate for the destruction done, nor can I conceive how free inspection of the sinus, to compare with that obtainable from the external operation, can be gained by it. A supraorbital extension of the sinus (recessus paracribrosus) could not be inspected from any intranasal opening, no matter how wide. Halle's operation is unnecessarily radical for the usual case of chronic frontal sinuitis, but may prove to be of value where unusual freedom of access, for instance for the removal of polypi from the sinus, is needed.

Drainage Tube. In most cases the channel into the sinus made by removal of the anterior ethmoid cells is so large and so well lined from the remnants of mucous membrane left by the curette

that it stays permanently open. There are cases, however, where cicatricial contraction of the mucous membrane about the outlet of the new channel into the nose or the formation of exuberant granulations in the channel itself will rapidly narrow it so that after a few weeks even a fine probe may pass with difficulty or not at all. These conditions are especially apt to occur where the ethmoid cells have been removed at some previous operation and have been replaced by hard thick bone, so that most of the enlarging of the ostium has had to be done forward by cutting away the internal nasal crest, with resulting denudation of hard bone. Such cases require the introduction of a drainage tube to keep the channel open until it becomes lined with epithelium.

E. F. Ingals was the first to introduce a drain into the sinus intranasally, employing his well known gold tube. The Ingals tubes are expensive and I have found them hard to introduce because their uniform curve does not fit the diverse anatomical conditions met with. For this reason I have devised the self retaining rubber drainage tube shown in Figure 7, h. It depends upon two rubber flanges to hold it in the sinus after its introduction, just as a self retaining catheter is held in the bladder. This tube is stretched over the introducer, Fig. 7, i), lubricated with a glycerin gelatine lubricant and passed into the sinus in the manner of a probe, the operator feeling a slight slip when the flanges have passed into the sinus. The tube is then cut off in the nose, about an inch and a quarter being left hanging down into the nasal cavity. A size of tube should be chosen that will enter easily and slip up and down in the channel when moved with light forceps. The tube must not fit tightly lest it cause headache and pain felt in the corner of the eye. It should be replaced by another in six weeks and by that time it may be possible to easily pass a larger size.

After-treatment. If a permanent opening of good caliber be established no after-treatment is needed in the usual case, not even washing out the sinus.

If a tube be worn, it and its neighborhood must be cleansed from time to time of adherent dried pus or mucus. The amount of this soon gets less. Oily applications to prevent scabbing can not be used without risking softening of the end of the tube in the nose. Where it seems advisable, the sinus may be washed out through the tube by means of a syringe. The tubes show no tendency to come out of the sinus.

If drainage be inadequate, suppuration will continue in spite of washing, sufficient drainage will cause the suppuration to cease unless there are irremediable pathologic changes in the sinus.

It is to be considered that the frontal sinus can not be opened so widely intranasally in proportion to its often great size as can the maxillary antrum in the lower meatus and that, while the artificial opening plus the natural outlet give the antrum through drainage, the frontal sinus had no such advantage when opened intranasally, but, having but one outlet, is in the state of an inverted bottle from which fluids flow with difficulty. For these reasons, therefore, minor secondary operations to restore or increase the caliber of the channel gained may be needed in some cases and the drainage tube may prove necessary where at first it seemed needless.

Williams advised passing bougies into the sinus after the operation to prevent adhesions. The channel is exceedingly sensitive for the first two or three weeks after the operation and even the introduction of a probe may create a severe headache. In addition, I have found that the attempt to prevent adhesions by separation in the healing process only creates irritation and is ineffective. I find it best to wait until cicatrization is complete and then to remove the scar tissue when all post-operative reaction has subsided.

Summary. The frontal sinus outlet or ostium is bounded in front by the crista nasalis interna, internally by the expanded portion of the anterior superior nasal spine of the frontal bone, externally by ethmoid cells lying between the ostium and the lachrymal bone, posteriorly by the foremost of the row of anterior ethmoidal cells. These cells are covered and completed above by the orbital process of the frontal bone which separates them from the cranial cavity. They form the portion of the sinus floor behind and external to the ostium and their removal gives, in most cases, ample space for drainage, creates a permanent channel into the nose and is preferable to merely cutting forward with rasp or burr to remove the internal nasal crest. The latter procedure is sometimes needed, in addition to removal of the anterior ethmoid cells, to help create a maximal opening. The ethmoid cells to be removed lie between the lamina papyracea of the ethmoid bone and its turbinal plate and are roofed by the orbital process of the frontal bone, as mentioned.

The operation usually begins with the severing of the anterior attachment of the middle turbinate, or with resection of its anterior half. In some cases the middle turbinate may be left intact. If needed the uncinate process is next cut away to expose the bulla ethmoidalis fully to view. The bulla is then entered from its lowest part with a ring curette whose edge is directed obliquely upward,

outward and forward and which is made to sweep away the anterior ethmoid cells from the bulla forward and upward to the ascending process of the superior maxillary bone, and, if possible, through the sinus floor up into the sinus behind the crista nasalis interna. If the sinus floor prove too hard to penetrate with the ethmoid curette an especially devised probe curette is passed through the sinus ostium, after the way through it has been found by an ordinary probe, and this probe curette is made to cut its way out of the sinus through the ethmoid cells under the orbital process of the frontal bone, thus enlarging the ostium enough posteriorly for the introduction of a larger curette of the same form into the sinus to clear away all of the cell remnants under the orbital plate. If necessary, then the ostium is also enlarged forward by cutting away the crista nasalis interna with a straight burr driven by the dental engine. The preference, however, is given to posterior enlargement alone because of the tendency to cicatricial closure of an opening into the sinus made in the forward direction.

In cases where there is reason to expect such closure one of the author's selfretaining intranasal rubber frontal sinus drain tubes should be placed in the sinus.

In cases where, with the sinus open for drainage, marked suppuration continues after the intranasal operation, the external operation must be resorted to. Where great swelling of the lids, exophthalmos or cerebral symptoms indicate the existence of caries of the sinus wall and progress of the disease beyond it in the form of Killian's sinuitis frontalis exulcerans the intranasal operation should not be done.

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THE ETIOLOGY OF PANSINUSITIS.*

Dr. J. Gordon Wilson, Chicago.

The thoughts which I venture to present on the etiology of pansinusitis are not new but represent my present attitude in regard to this subject. This has been to some extent the result of a series of observations on the effects of toxins in the frontal sinus which I began some years ago and which later have been extended by two of my colleagues in the clinic. We are not prepared at this time to publish our results. The work has proved much more complex than we anticipated and is far from complete. Most of our results have been negative yet some afford a basis for the suggestions I present in this paper.

One object we had in view in the investigation was to ascertain the possibility of local irritants of bacterial origin in one sinus setting up irritation in a neighboring sinus, healthy or aseptically injured either by direct continuity or diffusion of the irritants. We prepared capsules containing virulent micro-organisms which were inserted into the frontal sinuses of dogs. Both frontal sinuses were opened under aseptic conditions; one sinus was used as control and the following substances were placed in the other sinuses: celloidin capsules containing sterile bouillon, trypan blue, cultures of streptococci, staphylococci, bacillus coli, bacillus ozenae, and an unknown bacillus taken from a nasal discharge of a dog. As is known, such capsules permit toxins to pass through but prevent the exit of the imprisoned organisms. On one occasion there was introduced a cloth sack containing killed tubercle bacilli; on another, two cc., of a bouillon culture of a bacillus obtained from the nasal discharge of a dog. In all, care was taken not to injure the ostium and on post-mortem examination, the ostium was found patent though at times surrounded by swollen mucous membrane. The dogs were killed at periods of from forty-eight hours to five weeks.

The sinuses containing the capsules all showed marked reactions. They contained fluid of a serous, sero-mucous, or mucopurulent character but small in amount. The bacteriological examination is incomplete; we can only say that smears showed no organisms. The mucous membrane was thickened and if examined

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soon after the operation, was dark red in color. In those examined after long periods, the mucous membrane was thickened, but pale and edematous in appearance. The gelatinous appearance of the mucous membrane was at times very marked, in one case appearing like a polypoid growth. Microscopic examination of the membranes showed numerous round cells, especially plasma cells.

The sinuses in which no capsules were placed, with one exception appeared to be normal after the lapse of a few days required for the traumatic effect to pass. The sinus in which bouillon culture of the bacillus from a dog's nose had been placed had a normal appearance microscopically.

It seems that a frontal sinus is able to recover very well after opening, but after the introduction of the capsules and especially when these contained a diffusible irritant, there was a marked disturbance and permanent injury. While marked hyperemia was found in the other nasal cavities after the operation, in no case was this more than a passing effect. In short, no effects attributable to the toxins were ever found in adjoining sinuses.

The sinuses accessory to the nasal cavity can be anatomically grouped into two divisions:

(1) An anterior, comprising the frontal, maxillary and anterior ethmoidal, which opens into the middle meatus beneath the middle turbinate.

(2) A posterior, comprising the sphenoidal and posterior ethmoidal which opens into the superior meatus above the middle turbinate.

A pan-sinusitis may involve either of these groups or both.

In discussing the etiology of pan-sinusitis it cannot be your wish and it is not my intention to enumerate the diseases in which it has been found to occur or to tabulate the micro-organisms that may be the apparent aggressors. This has been amply covered by numerous articles and by many text books. It is rather my intention to consider some local anatomical and physiological factors which explain why pan-sinusitis does not occur more frequently and, which when defective are destroyed, lead to its occurrence and persistence. I feel that you may justly blame me for dwelling on many elementary facts. My apology is that elementary facts are the basis of knowledge. I believe that it is good to reconsider from time to time nature's method of preventing disease and of effecting spontaneous cures. Such a reconsideration strengthens our desire to safeguard with all jealousy the barriers that have been erected for man's protection.

There are some elementary facts which afford a basis from which one can start—so well recognized that one need but state them:

(1) No sinus inflammation can occur unless the physiological activity of the sinus be disturbed or destroyed.

(2) It is not the physical presence of microbes either in the sinus or its vascular system that as a rule induce inflammation, but the toxins which come from their growth and disintegration. The condition of the tissue on which the bacteria are implanted influences the future course of the disease.

(3) The low grade of inflammation which we associate with ordinary catarrhal colds may have as its primary cause irritants other than those which arise from the local presence of pathogenic organisms. Under this would come climatic disturbances and metabolic disturbances due to disease elsewhere in the organism. By a catarrhal inflammation I understand a slight grade of change, affecting mucous membranes, whose dominant feature is irritation rather than necrosis. The irritation is shown by the congestion of the underlying vessels, abundant exudation of serum containing some leukocytes and the casting off of many of the columnar epithelial cells. The excretion of mucous is increased and forms a layer over the inflamed surface. This mucous discharge is, to a considerable extent, protective; not only are bacteria detained in it, but it is claimed to have a certain amount of bacterial activity, and irritants which may appear on the surface during the attack diffuse through it with difficulty.

(4) The lowered resistance which occurs in general metabolic disturbances (e. g. diabetes) may produce more chronic and insidious processes, but the part played by them in disease apart from organisms is not known.

Passing from these generally accepted principles we may now consider some of the anatomical and physiological factors present in the sinuses whose structure and activities prevent the occurrence of disease. From this point of view I would ask you to consider the ciliated cell and the lymphatic system. I need not remind you that the mucous membrane is lined by ciliated cells with numerous mucous secreting cells.

In the ciliated cells the cilia which are very fine and relatively short are extensions of cell protoplasm covered like the cell with a delicate membrane. Their action consists in a simple bending over of all the cilia which crown the cell, followed by their return to the vertical position. Their purpose in the sinuses seems to be

to produce a movement of fluid or mucous over the surface which they cover in the direction of the ostia, and working in harmony with the nasal cilia the secreted mucous is moved on towards the anterior nares. The secreted mucous gathers up all extraneous matter which is cast off into the sinus cavity during the life of the cell and any extraneous particles which may have accidently entered during the diffusion which results from the positive and negative pressure present in the sinuses during respiration.

"During the life of the ciliated cell the cilia are in constant activity bending over and recovering themselves many times a second. So rapid is the movement that the action of the individual cilia cannot be seen except when it begins to slacken, which may be caused by cooling the preparation or may accompany commencing death of the cells. But the general effect of the whole movement as it has been studied elsewhere has been compared to the rhythmic wave-like motion which is produced by the wind upon a cornfield."[1] The movement begins at one part of the ciliated surface, and gradually traverses the whole surface in the form of a wave, and this is succeeded by others at regular intervals. The decrease of the ciliary movement is at least partly responsible for the invasion of the sinus by bacteria just before death in some cases and after death in all cases. The normal ciliated mucous membrane is impervious to micro-organisms. If the surface be not too greatly destroyed or denuded during the course of the inflammation or operation the epithelium which grows in from the side as flattened epithelium ultimately acquires cilia, the amount of regeneration being influenced by the age and condition of the patient.

The arrangement of the lymphatics of the large sinuses has a direct bearing on the liability to infection. The elaborate interlacing vessels in each sinus radiate towards the ostium where they surround its free border. They there communicate with the main channels in the nose where they pass back into the depression between the tube and the inferior turbinate. The direction of the lymph flow in each sinus, therefore, diminishes the tendency to lymphatic infection.

It will be apparent from this brief sketch that we have in each sinus an anatomical and physiological mechanism of simple type adapted to free these cavities from the accumulation of such local irritants as arise during the normal life of the cells which line it, and an isolated lymph system giving the best possible chance against microbic invasion. With this in mind we may proceed to consider

the changes which result in the breaking down of these safeguards.

Thus far we have dwelt on the independence of the sinuses in regard to their lymphatic system and their ciliary mechanism. We now must recall that these cavities are largely supplied by a common artery (the spheno-palatine) and are closely associated with a common vasomotor mechanism at the upper part of the respiratory tract and that the ostia in each division are relatively close to each other. In short, that an irritative lesion of even moderate intensity, in or near one cavity, must affect the other cavities. more or less. This was shown in our observations on aseptic traumatic injury of the frontal sinus in dogs.

From this standpoint it will be well here to consider how that most common vasomotor disturbance arising from a variety of causes and generally termed a rhinitis will affect the sinuses. In ordinary rhinitis we have a local inflammation, associated with it we have a congestion of the whole nasal mucosa and submucosa. This congestion involves more or less all the sinuses and especially those in one group. Into them mucous is discharged but the epithelial structure is still intact, though some shedding of the epithelium comes away with the exudate. The openings, though restricted in size from the swelling are not undefended by the cilia and mucous. On the whole there is little chance of invasion of discharge or of bacteria directly from the nose. As the local inflammation subsides the congestion disappears, the cilia rapidly recover and the exudate which is not absorbed is cast out by the ciliary movements and the sinus recovers. If, however, the inflammation be so acute that the physiological protectors and barriers are interfered with or no longer effective the morbid processes are different.

(a) The cilia no longer can remove the effusion which accumulates and this is increased by the action of gravity in such a cavity as the maxillary. This mucous accumulation with its epithelial debris acts as a mechanical irritant to keep up the sinus engorgement. From the formation of some of the cavities it is obvious that this secretion may persist after the rhinitis has disappeared.

(b) The nasal lymphatic engorgement present in all inflammations may be such that the disturbances in lymphatic circulation in the congested sinus produces a lymph stasis and the initial step in a lymphatic infection, or the presence of the accumulated fluid referred to above and the persistent congestion may at a later stage produce the lymph stasis and infection.

(c) Following the lymphatic disturbance come bacterial invasion, either by direct extension along the lymphatics or by the infection of the exudation from the lymphatics, and the sinusitis. Luckily, although all the sinuses are involved in the congestion, the resulting suppuration in all or even in one is relatively rare.

(d) In some cases it may be that we have a mechanical interference with the cilia by bacteria similar to that found by Mallory. In such there would be the possibility of superficial invasion by micro-organisms.

Chronic pan-sinusitis, the form in which the disease usually comes to our notice, appears to me to follow the same general course, namely, a failure in the cilia to remove effectively the secretion, a chronic turgescence resulting in repeated lymphatic stasis and finally bacterial invasion. In some cases the cause may be a slight chronic rhinitis with mild exacerbations or the involvement of one sinus with recurring subacute attacks of rhinitis with gradual involvement of the other sinuses. As we shall point out later it is exactly in such cases that micro-organisms abound.

Other factors at work may be, for instance:

(a) Narrowing or obstruction of the ostia or of the nasal cavity from mechanical causes.

(b) Disease in the adjacent part of the nasal cavity which has resulted in destruction of the cilia.

(c) Scar tissue, tissue devoid of cilia, either from disease or from nasal operation.

The chronic turgescence may be so slight that it is beyond our diagnostic ken and never awaken suspicion. It may be so slight that bacterial activities and toxin effects are counterbalanced by tissue activities or antibodies within the limits of health. It is in such cases that a slight increase in congestion may be enough to disturb the balance. It is in such cases that debilitating diseases and hematogenous infections may set up a pan-sinusitis.

So far I have said nothing about the particular variety of infection. My idea has been to get at the general principles which underlie all. When we consider the bacteria involved we find it satisfactorily established that a variety of micro-organisms may be found in the healthy nasal and postnasal cavities of most individuals. These bacteria are frequently few in numbers, of low vigor, and of little or no virulence. In the sinuses of man the evidence available compels one to accept the view that the normal sinus—certainly the frontal and maxillary sinuses—contain no bacteria. On the other hand, in the nasal cavities and probably in

the accessory nasal sinuses of many persons who are apparently in the enjoyment of good health, but who are at the same time suffering from a chronic catarrhal inflammation of the mucous membrane of their upper respiratory passages, bacteria are present and are more or less virulent. From a variety of causes which diminish the tissue resistance such as debility, chills, traumatism, their virulence is increased. It is in such cases that the introduction of a fresh organism may produce changes which allow bacteria already present but quiescent or avirulent to become virulent and give rise to acute or subacute infections. It is along this line that a gastro-intestinal disturbance produces such metabolic changes that it may set up a coryza and the treatment call for a recognition of this etiological factor. The chief organisms which are found and which demand our attention are the pyogenic cocci, the pneumococcus, the micrococcus catarrhalis, the influenza bacillus, the meningococcus and the bacillus tuberculosis.[2]

It may be argued that I have not considered many etiological factors which influence our treatment. Thus there is the seasonal factor. In winter we have the respiratory attacks predominating, in summer the gastro-intestinal, and in the transitional period the respiratory and gastro-intestinal. We have the former period when colds and coryza predominate, the second period that of diarrhoea and food intoxications, with coryza secondary, and the period when both are equally at work. In our treatment we have to consider these etiological factors, but from the standpoint of this discussion these are secondary to the general principles to which I have referred.

It may be argued that I have paid too little attention to the specific organism. This is true because I believe the organism is most often an invader into an already damaged sinus. This appears to me to have a bearing on the question of the results of focal infections. And further, the same morphological organism can vary so much in its virulence that until we know more about the cause of this variation we are not likely to get far in considering its etiological significance.

An interesting etiological speculation might be the antitryptic property of exudate into the sinus. Serum normally is bactericidal. On standing or in the presence of leukocytes undergoing decomposition it loses this property, due, perhaps, to the loss of its antitryptic property. It then becomes tryptic and acts both as an irritant and a suitable medium for growth. Whether it has any influence here we do not know. It is at present interesting because

of the recent suggestion of Almroth Wright that wounds can be irrigated by antritryptic serum.

It may be urged that I have neglected to enumerate such obvious etiological factors of pan-sinusitis as the overflow of secretion from one sinus into the other; as the possibilities of defect in the septal walls tending to its causation and many others. That is true; I have endeavored to leave alone individual facts and get at general principles.

In conclusion, I would express the opinion that a healthy sinus has great protective power against bacterial invasion and great recuperative power after the simple hyperemias which occur during a rhinitis. I would urge you to consider that it is chiefly in the chronic rhinitis that we have the potentialities of a pan-sinusitis. These potentialities are influenced by:

(1) Circulatory disturbances, vascular and lymphatic, accompanying or produced by the rhinitis.

(2) The inability of the sinuses to clear themselves of the excess of exudate, resulting from the inflammatory irritation.

(3) The infection.

The disturbance resulting from the first is such that there follows the failure of the second which inevitably leads to the invasion of bacteria. I agree with Wright and Smith[3] that "even in the most virulent cases, with high temperature and the systemic signs of sepsis, there is unquestionably an antecedent change in the mucous membrane and its secretions which have afforded these frequent denizens of healthy noses an opportunity for maleficient activities. A severe coryza terminating in resolution and a restoration of the structure of the intranasal mucosa to a fairly normal condition often takes place when the mucous membrane of the accessory cavities remains charged with the products of inflammation." I ask your special consideration to this last sentence as I believe we do not realize its full significance. Our best internists appear more alive than some rhinologists to the possibilities of the accessory sinuses being charged by the products of a low grade inflammation--producing apparently little or no local observable effects. The time has come when we are being asked if we have not in the sinuses the possibility not of a suppuration cavity but of a cavity where endo-toxins are being set free, resulting in an auto-intoxication.

It is our task to diagnose and treat. But the wise treatment will recognize why nature has broken down and aim as far as possible at compensation. It will aim at not leaving any one sinus

so impaired that it acts as a source of danger. To replace epithelium in a dependent cavity by scar tissue is to replace it by tissue with no physiological action. I cannot conceive of a healthy antrum ever existing after extensive curettage. The teaching of nature is obvious. The mucous membrane of the accessory sinus is doing important work. We may aid by reducing chronic vascular and lymphatic engorgement. We may aid by assisting in the removal of excess of secretion. But if we do so at the expense of permanent damage to the ciliated walls we may well go slowly, and ask what compensation we offer for this loss.

Our knowledge of the etiology is unsatisfactory; our knowledge of the cause and effects of low grade inflammations most disappointing. But it is along the lines which I have sketched that some day we hope to know more.

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Operations for Clefts of the Hard and Soft Palate. L. EMERSON, *Jour. A. M. A.*, Jan. 23, 1915.

Owing to the mortality when the Brophy operation, in the hands of the occasional operator, is performed in very early infancy, Emerson has abandoned the early operation entirely. If complicated by hare-lip, he usually operates on the hare-lip during the first few months and upon the palate some time during the second year. Unless the cleft palate is remedied within the first five years of life, the power of speech will be seriously and permanently impaired.

Emerson's method is to remove a wedge-shaped piece, base down, subperiosteally from the septum, and the space thus left is obliterated by forcing back the premaxilla, leaving a septum smooth on both sides. The edges of the premaxilla should be freshened as well as the maxilla, in the same way as in the case of a single cleft. The technic is illustrated fully and described at length. Ed.

NON-OPERATIVE TREATMENT OF THE ACCESSORY SINUSES.*

DR. LEWIS A. COFFIN, New York.

In the light of a fairly long experience, and in the light of the present-day laboratory teachings, I have ceased to think of the cure of the disease of the accessory cavities of the nose, by either operative or non-operative procedure. I am satisfied when I have accomplished a result sufficient in any case that I may speak of the disease as arrested.

My experience has been that in many cases in which I have done most thorough and radical operations there have been recurrences of the disease at variable lengths of time. The time has varied from one to five or six years. Another matter of experience is this: A sinus diseased and treated to the point at which the patient is discharged as cured seems more liable to a re-infection than do those sinuses in the same patient to become diseased which have not previously been affected. We have analogies in other sections and organs of the body and in other diseases. One man may have more or less frequent attacks of boils always occurring in the same location on the body, whereas he is apparently well between attacks. I know a man who has periodically a carbuncle, always at the same site.

Syphilis and tuberculosis in their recurrent manifestations teach the same lesson. Perhaps we may explain the matter in present-day language, thus: if we consider the infecting germ as an invading army, we witness the efforts of the invaded host as those of an army which enters on a defensive warfare. The manifestations of this warfare constitute disease and the result of battle is determined according to the relative strength of the combatants. As physicians, we array ourselves on the side of the defense and it seems that we may hope to vanquish the invading army somewhat according to the method and cunning of his entrenchment. Our efforts are twofold; first, we endeavor to destroy the enemy and, second, we try to so bolster up and strengthen the invaded body as to enable it not only itself to put up a better fight, but to the end that it may come out of the conflict in better condition than it could otherwise have done.

* Read before The American Laryngological Association, Niagara Falls, Can., June 1, 2, 3, 1915.

It would seem that at a certain point when the invading army has been so effectively overpowered that all signs of disturbance cease there may remain many of the invaders, and that through entrenchment on the one hand or effective interment on the other, warfare ceases and the body is at peace, until such time as for some reason the balance of power between these elements is again in a state of unstable equilibrium and the manifestations of renewed strife appear, or medically, a relapse has taken place.

Undoubtedly it is scientifically possible and parctically demonstrable that cure does occur, but it is not scientifically nor practically possible to say at the time of the particular patient's discharge from further treatment whether the arrest of the disease is temporary or permanent; but I am wandering a-field.

My purpose at this time is to discuss the treatment of sub-acute and chronic empyema of the accessory cavities of the nose along non-operative lines, the treatment of which, further than the mere toilet of the nose has been essentially surgical.

So well established is the treatment of acute sinusitis that I shall not take the time of this society in discussing it.

Of the non-operative treatment of the more chronic forms of sinusitis, however, I have given much thought during the past year and am glad to feel that my efforts have not been without some degree of success. I purpose to outline the development of the method I am now using, describing the apparatus, medicinal agents used and report a few cases.

During the winter of 1913-14 I was treating sub-acute and chronic empyema of the accessory cavities of the nose by the application of negative pressue in conjunction with autogenous vaccination. The negative pressure was obtained in the following manner: into the nostril on the side of the head to be treated was inserted an olive-pointed tip connected by rubber tubing with a vacuum bottle and sufficiently large to close the nostril; the ala of the other nostril was pressed against the septum and the patient instructed to say, ka, ka, ka, while the suction was applied. This procedure does not always pull the mucus and pus into the bottle, in fact it never pulls all the muco-purulent secretion into the bottle, but this can be accomplished subsequently by means of a canula connected with the suction apparatus. Or, after as much muco-purulent material has been drawn into the nose from the accessory cavities as is possible by the suction, the nose may be cleaned by a nasal douche. But one case of empyema was completely arrested by this method. This was a case in which the disease was confined to those cavities which drain into the superior meatus.

One day during the early part of the winter just past I took to the pathologist of our hospital a culture taken from high up in the middle meatus of the nose of a patient on whom I had just previous to taking the culture made several applications of the negative pressure. The pathologist said he did not think it possible to pick out the pathogenic germ as the plate was so densely covered by air germs. Someone standing by remarked that this was probably on account of the in-rush of air following the removal of the nasal tip of the suction apparatus. To myself I thought if this be so, why may we not offer a medicated air to these vacuumized cavities? I immediately constructed an apparatus by means of which I was able to do this.

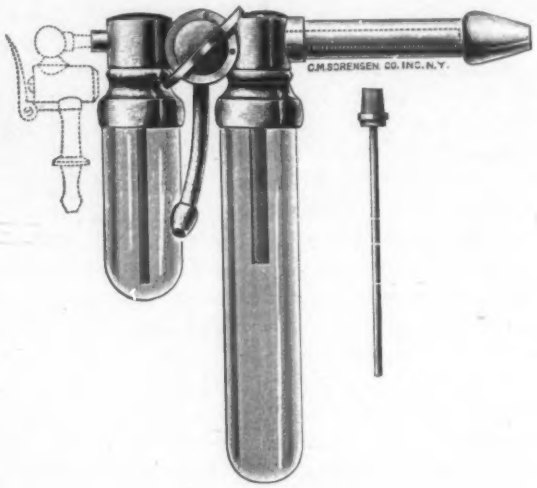
The air entering the vacuumized nose and cavities under a considerable pressure is medicated by a nebula of oil variously laden with remedial agents. The vacuum bottle and the tube leading from it to the nose are open to the pressure current through the nose when the exhaust pump is shut off from the vacuum bottle. This permits the pressure current to drive the mucus and pus from the nose into the vacuum bottle. The change in the nose from a positive to a negative pressure may be made as often and for as great a length of time as one desires, or as the case demands. By means of this procedure one may draw from the accessory cavities much more pus than can be done by suction alone, and to my mind there can be no question but that the medicated air reaches well into these cavities. I have used autogenous vaccines in conjunction with this procedure and have been able in two chronic cases to cause an entire cessation of discharge in which suction alone in conjunction with the vaccines had failed entirely. It sometimes happens that even with the combined use of the negative and positive pressure a certain amount of secretion cannot be drawn or swept into the vacuum bottle, for that reason. I have a small silver catheter which attaches to the nose-piece of the instrument. With this catheter one can pick up any residue of secretion from any part of the nose. After the nose and air cavities have been as thoroughly cleansed as possible the process of alternate suction and pressure is done from 10 to 20 times in order to carry the modified air well into the cavities. Both the negative and positive pressure are under control and the amount of each is registered by an index.

If the negative pressure be started low, say at 5 units,† it may be gradually increased to 15 or 20 without discomfort to the patient.

† A unit is such a pressure as will support a column of mercury one inch in height.

The nebula is thrown into the nose under a pressure of from 10 to 15 units. Cases of atrophic rhinitis can stand neither a negative nor positive pressure with comfort beyond about 8 units.

The instrument consists of two (2) bottles mounted on the lower side of a tube one-half (½) inch in diameter, which I call the connecting tube. The bottles are mounted close together at one end of the tube, the other end of which terminates in an olive-pointed tip about three and one-half (3½) inches from the bottles. Between the bottles is a switch key by means of which the inside of either bottle may be connected with the lumen of the connecting tube, the inside of the other bottle being at the same time disconnected from the same. Between the bottles also is a small tube extending through the key sleeve for the attachment of rubber tubing which connects the apparatus with an exhaust pump. When the key is so turned as to connect the second bottle with the connecting tube one is able to create a vacuum in this bottle by working the exhaust pump. This bottle is five inches in length and one inch in diameter and is the vacuum bottle of the apparatus. This bottle connects with the nose by means of the olive point and when the naso-pharynx is closed off from the oro-pharynx the nasal chamber and any cavities connecting with it are partially vacuumized when such a condition exists in the bottle. The nasal chamber may be



closed either by instructing the patient to say, ka, ka, ka, or the ballooning of a small rubber bulb in the chona.

The first bottle is a nebulizing outfit; it is three inches in length and one inch in diameter and may be connected at the proximal end of the connecting tube by means of an ordinary shut-off with a pressure pump or tank. It is the medicine bottle. When the shut-off is open a nebula is formed in the bottle and if the key be so turned as to connect this bottle with the lumen of the connecting tube the nebula is thrown into the nose through a small tube inside the connecting tube. If the key is not so turned the nebula is thrown from a safety vent between the bottles. By working both the suction and pressure currents at the same time one may produce in the nose either a suction or a compression effect at once and may alternate from one to the other immediately by turning the switch valve between the bottles.

Remedial Agents: Whereas the apparatus presented may be so constructed as to throw into the vacuumized nose a liquid spray, nebula or vapor, the one which I have used is so constructed as to throw either nebulae or vapors. I first used oxygen, but, subsequently, have been using mineral oil, variously medicated, as a nebula. My first combination was a 5 per cent solution of carbolic acid and Churchill's tincture of iodin. In this preparation the iodin seemed to be thrown out of solution by the acting of the nebulizer. I am now using either an oil loaded with Bulgarian bacilli or one of the proprietary preparations of iodin. I have used both liquid Iodex and the 5 per cent solution of Iodin Petrogen.

Case Reports: Mrs. J. W., a lady 77 years of age, consulted me on October 1, 1914, on account of a copious discharge from the left nostril. She came with her own diagnosis of sinus disease, as she said she had been treated all the previous winter for the same trouble, which had originally been diagnosed by her oculist whom she had consulted on account of the pain in and above the eye. At the same time she came to my office she told me that at home, on account of the discharge, instead of a handkerchief, she was using soft paper and burning it. I treated her daily for two or three months by means of suction and vaccines. She was kept more comfortable during this time, but there was a little difference in the daily output of the muco-purulent discharge. The vaccines were stopped—while the other treatment continued. I began the combined use of suction and compression about Jan. 15, 1915, using the iodin and carbolic nebula under considerable pressure. By this method I for a considerable time obtained a much greater quantity

of the muco-purulent secretion. About the last of February she asked if I would not again use the vaccines as she was sure she felt better while they were being used. The vaccines were accordingly prepared, but about this time, to be exact, on the 2nd of March, she stated that she should not return for further treatment. On the 3rd of March I received my first consignment of oil loaded with the Bulgarian bacilli. As my patient had based her determination not to return on a financial basis, I felt at liberty to write and ask her to return and offered to treat her without money consideration. She accepted the proposition and on March 5th I resumed treatment, using a nebula of oil containing the Bulgarian bacilli and again began the vaccinations.

Previous to this I had two or three times a week irrigated the antrum, always washing out a considerable amount of pus—this process was not repeated after beginning the use of the Bulgarian bacilli.

Improvement was marked, and rapid. No pus was seen in the nose after the 20th of the month, and on the 24th she showed me a perfectly clean handkerchief, which she had carried for two (2) days. I have the handkerchief. I continued the treatment at from intervals of 2 to 4 days for about two weeks when she was discharged in a condition which she described as "absolutely cured."

Case 2: An interesting case is that of a young Polish woman, J. A., 20 years of age, who came to the eye clinic of the Manhattan Eye, Ear and Throat Hospital on January 7, 1915.

She gave a history of supra-orbital headache of more than a year's duration.

Diagnosis of episcleritis was first made, later a cyclitis developed. Patient complained of intense pain. The treatment consisted of leeches, atropin, hot fomentations, asperin and mercurial innunctions, all of no avail. She received one injection of tuberculin for the scleritis which became worse immediately afterward—I was asked to see her on January 21, 1915. Eye was swollen, partially closed on account of a droop of upper lid, tissues about the eye were almost mahogany-colored, nose filled with pus.

She complained of great pain in the eye and at the lower and outer border of orbit. Was extremely tender over all palpable sinuses, could speak but few words of English and shrank from all painful procedures.

I advised a radical Killian operation and appointed time for same. At this point her brother made appearance and objected to any op-

eration which might leave a scar if it could possibly be avoided and asked that Dr. MacKenty be asked to see the case with me. Dr. MacKenty agreed with me that probably the Killian was the best procedure, especially as, on account of a considerable bowing of the septum to the diseased side, good intranasal operation could not be done. The brother consented reluctantly. During the two or three days that elapsed before the operation was to be done I determined to see what I could do intranasally. I removed the anterior end of the middle turbinal, put her on douches and applied suction. She improved as to pain and eye symptoms.

About this time I had my apparatus working and determined to test it out on this case. When she had been under treatment for two weeks, all pain had ceased; the eye was normal and she returned to work. We were still getting great amounts of pus from the nose. Vaccination was done while she was still in the hospital. The streptococcos in pure culture was the infecting agent. The secretion of pus began soon to diminish and I have not seen pus in her nose now for a period of about two weeks nor have I been able to draw it into the vacuum bottle.

Case 3: Miss S., age 22, has been catarrhal for several years. Three years ago double mastoiditis—operation—referred to me again on January 19, 1915, on account of frontal headache following an attack of grippe; pus in middle meatus of left side; tenderness over left frontal and septum much bowed to same side. Treatment by alternate suction and compression begun at once. Headache gone after first treatment. No pus in nose after ten daily treatments. This was an acute case and equally as good results might have followed other forms of treatment.

Case 4: Mrs. S., age 48. Did a radical Killian on Mrs. S. eight years ago for the relief of a chronic pan-sinusitis of the left side. A Caldwell-Luc operation was done on antrum at the same time, operation followed by cessation of pain and general constitutional improvements. Discharge never entirely ceased. Douching, spraying and such other procedures as can be done by a patient in her daily toilet were practiced by Mrs. S., and occasionally the various sinuses were treated at my office. She came for such a course of treatment on March 30, 1915, complaining of an increased nasal discharge and a troublesome cough. I began the alternate suction and compression treatment at once, together with autogenous vaccination. By this process the pus and muco-purulent secretion which could be gathered into the vacuum bottle measured at least one inch in height

in that bottle. Cough was lessened after first treatment. Constitutional improvement was soon evident and secretion gradually diminished until to-day, May 25th, I have been unable to get even a show of pus or mucus in my vacuum bottle. Treatment in this case will continue for two or three weeks. I am confident of the complete arrest of the disease.

Case 5: Mrs. H. M., age 35. Has had more or less trouble with her throat since childhood. I removed her tonsils and adenoids on December 7, 1914. No complications. Good recovery. Had complained for several years of pain, especially when tired, in and about the left eye. On January 25, 1915, she began seeing double. Was obliged to keep left eye closed.

I referred her to Dr. Herbert Wooten, who diagnosed a paralysis of certain of the eye muscles and advised treatment by bichloride of mercury. As this seemed to imply a specific cause for the eye symptoms, I hesitated as I knew her father, mother, husband and son and could not believe there was a specific taint.

A Wassermann was done on blood and spinal fluid—both negative. There was considerable indican in urine and the bowels were given a rigorous cleaning out, patient remaining in bed for several days. After four or five days of bowel treatment paralysis of the eye muscles disappeared. After several weeks in Florida she again consulted me. Had had no further diplopia, but the upper lid of the left eye drooped so that she complained that one eye was growing smaller. For some reason I applied suction to her nasal cavities. I was surprised to get a considerable amount of muco-pus from the left side of her head and more surprised on the following day to find both eyes wide open. The treatment above described was given four or five times, when she ceased to come to the office until she came last week with her son. Asked why she had not continued treatment, she answered, "Why, I'm all right; I can now sit through an entire movie performance without pain in my eye and I did not suppose you wanted to see me when I was all well."

These few cases will serve as types of sinus troubles that I have treated without operation. All I have treated are not well, but neither they nor I have abandoned hope of an ultimate arrest of the disease.

156 West 58th Street.

TECHNIC OF SUSPENSION LARYNGOSCOPY.*

DR. ROBERT C. LYNCH, New Orleans.

I assure you that it is an honor and a pleasure to be privileged to discuss the subject of suspension laryngoscopy with you, and I trust you will do me the honor to enter into the discussion freely that we may determine if possible the limitations and best technique of this new method of examination of the larynx.

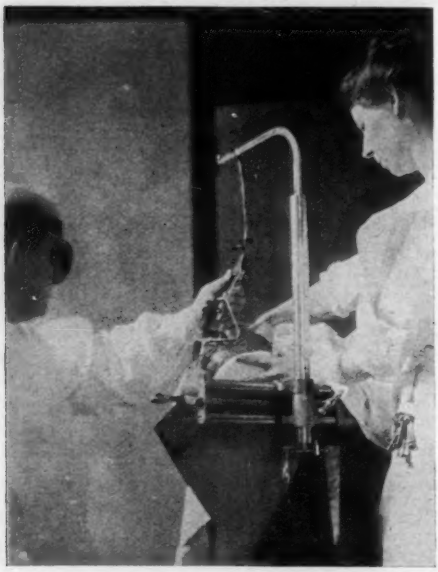
As an excuse to you for the presentation of a much altered or new model of the hook I would like to discuss with you the shortcomings of the older models I have worked with and of Prof. Killian's new models.

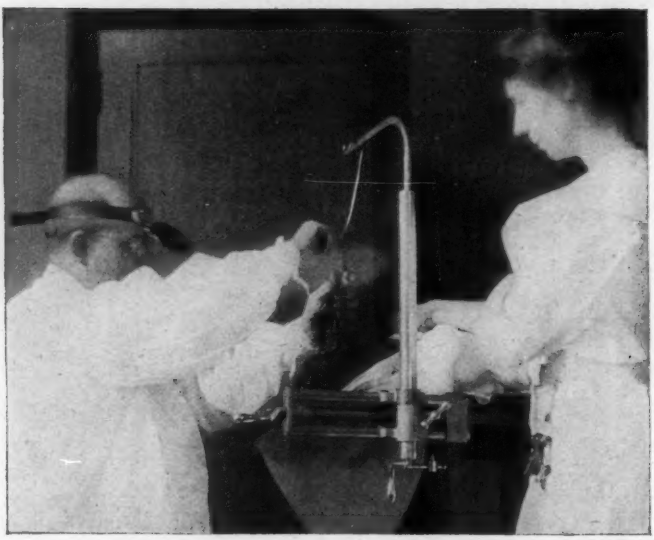
This original model, as you know it, had these disadvantages: First, the mouth gag only opens, as you see, to a scant one inch in the vertical; the tooth plate is small laterally and shallow vertically and has only the movement of one-fourth of an inch in the horizontal plane. Last year at Atlantic City I presented this modification of the tooth plate which opens a full inch and a half in the vertical and adjustment of nearly an inch in the horizontal plane by turning this milled screw. With this modification I worked for nearly a year with more satisfaction than with the original but not to my entire liking. It soon became apparent that the horizontal movement of the traveling crane was too limited so I had constructed the table top which Dr. Arrowsmith has incorporated in this table, permitting the placing of the crane at any distance on this horizontal projecting platform that will admit of at least ten inches more movement if necessary. I consider this table top absolutely necessary in its detail if you follow my technique in acquiring your view of the larynx under suspension.

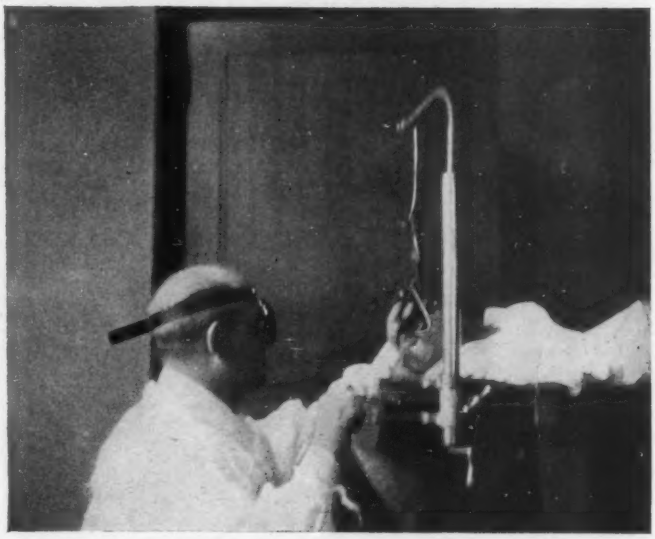
This new model I have to show you I have used about six weeks and so far it surmounts all of the difficulties that were noticed in the former models and has some features that are entirely new.

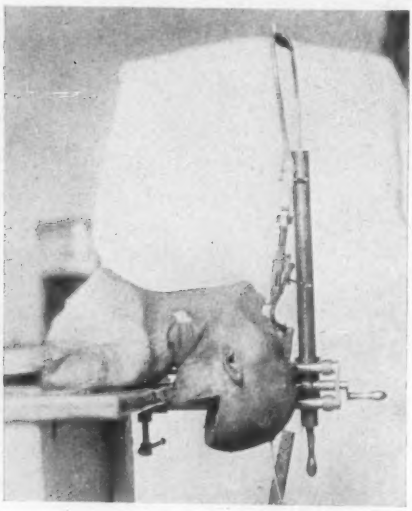
The instrument is constructed of steel that is very hard but is not brittle. While all of the old models were of brass, a brass instrument, even of this type, would be worse than useless. The base to which the tongue spatulas are attached is very heavy and withstand

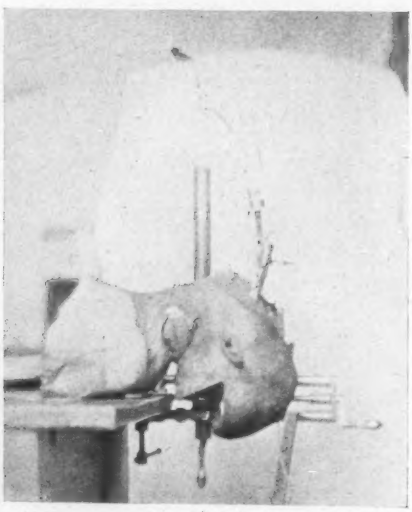
*Read before the Section on Laryngology and Rhinology of the New York Academy of Medicine, May 25, 1915.











far more strain than it would ever be necessary to employ. The body is heavy, too, it is true, but it is necessary to secure this large screw for the movement of the mouth gag in the vertical direction. It is also drilled throughout its circumference so that if by any possibility it should stick or jamb during its opening one might employ this small handle as one would a jack and screw and thus secure sufficient power to overcome this difficulty.

This new gag will now open a little over two inches, which is in excess of the widest mouth I have seen up to this time. The pear-shaped ring has many advantages over the old model: First, it is attached at one part of its circumference to the body of the instru-

ment and its movement is controlled by this screw which converts the ring with a very powerful lever; second, at an opposite point are placed one or two or even three tooth plates so that we can better distribute the pressure against the teeth and the tooth plates, as you see, are detachable and move at will to any point along this bar; third, I will call your attention to the most important feature of its movement, namely, that if the open jaw represents the fixed point and the tip of the spatula a moveable point the turning of this screw not only drives the spatula to any desired point on the laryngeal face of the epiglottis but also has this tilting action which tends to

elevate the epiglottis that it may better be brought into a sraight line with the open teeth, thus giving a broader view of the interior of the larynx. Then using two tooth plates as points of fixatim on the unmoveable upper jaw prevents the tongue from slipping from side to side on the spatula, a most annoying state of affairs. Lastly, I call your attention to the tongue spatulas which are black to prevent light reflection; they are rough on the tongue surface to prevent slipping and have these two fixed wings, which keep the tongue well out of the way and give the broadest possible view. That part of the tip which is in relation with the epiglottis forms an obtuse angle with the body of the spatula giving, I believe, one-quarter inch more view antero-posteriorly and securing a better view of the anterior commissure. Finally, I have added one more hook to the handle of the spatula that one may have more latitude in the vertical movement of the suspension. Thus, if the crane is extended vertically to its limit and the view is not good, it can be moved down about four inches, placed upon this second hook and you now have four inches more movement in the vertical direction.

I have added these two light attachments to be used for demonstration, and this long light carrier which I call a searcher long enough to pass into the trachea and search the side walls for any pathological condition. The technique I can best illustrate with lantern slides. No. 1 will show the position of patient and assistant holding the head slightly extended with chin in the middle line. No. 2, instrument closed, tooth plates adjusted properly, the hook bent moved to an acute angle so as to be out of the way of the arm of the assistant; the mouth opened, the tip of the tongue spatula follows along the post-pharyngeal wall until the tooth plates are fixed behind the teeth of the open jaw, being sure to keep the instrument in the middle line. No. 3, the mouth gag is now opened wide, which assures the tongue being held in the middle line and the tip of the spatula is driven down under the epiglottis by turning the screw on the pear-shaped ring; the traveling crane brought into position and the spatula hooked into place. No. 4, by turning the worm gear joint on the handle we begin to produce extension, on the spatula, and one now has a view of the hypopharynx and posterior half of the larynx.

No. 5, by moving the crane horizontally one increases the extension and gradually brings the interior of the larynx into view.

No. 6, then by producing more pressure by raising the crane vertically we again add to the view. No. 7, shows the patient completely suspended so to bring the anterior commissure into view and, if

you will pardon me, I will call this the Lynch position to distinguish it from No. 8, the Killian position, where the crane overlies the tip of the spatula and in which I cannot obtain the view I do working by my own method. No. 9, is a view of the interior of the larynx as shown in No. 7, the vocal cords are distinct and if they are presented to you in the manner I am sure you will agree with me that all intra-laryngeal manipulations are simplified and whatever may be attempted will be done far more accurately than by any other known method.

Dissection, suturing, etc., in fact, you are now prepared to treat the larynx in a truly surgical and scientific manner. I have removed nodules, single pedunculated and papillomatus tumors, multiple papilloma, intrinsic epithelioma, curetted, cauterized, intubated and removed specimens, I have sutured intra-laryngeally on two occasions, and in one instance produced a plastic flap.

1202 Broadway Street.

Operative Treatment of Cleft Palate. H. BLAKEWAY, *Lancet*, March 13, 1915.

Blakeway reviews the relative merits of the operation of "median suture" and "turn-over-flap" and concludes that the Langenbeck operation is still the best for routine use. The width of the cleft, the height of the palatal arch and the general health of the child have all to be considered before a decision is reached. If the condition is complicated by harelip closure of the latter during the first few weeks of life has a valuable effect in causing obliteration of the most anterior part of the cleft. The suggestion that the worst cases of cleft palate can only be closed by a flap operation in early infancy is difficult of decision. The expectation that on account of the early age at which the operation can be done the speech will be much better than in cases where operation has been deferred has not yet been realized. ED.

POST-TYPHOID ULCERATION AND STRICTURE OF THE ESOPHAGUS.*

DR. ROBERT L. MOORHEAD, Brooklyn, N. Y.

Ulcerations occurring in the course of typhoid fever have been demonstrated upon almost every mucous membrane of the body. Rarely has such a condition been found in the esophagus, and only slightly more frequent are the reports of cases of the resulting stricture formation in those patients so fortunate as to have survived the disease.

As early as 1834, Chomel[1] reported the case of a young man, aged 20 years, who died on the thirtieth day of the fever, and on autopsy, multiple ulcerations of the esophagus were found. In 1841, Louis[2] reported a series of 46 typhoid autopsies of which 7 presented ulcers of the esophagus, but the reason for this large percentage is not apparent and is the more remarkable when one considers that few more than this number have been observed in many thousand autopsies since that time. In this series, the ulcerations were found most frequently in the lower end of the esophagus, and in three cases the ulceration involved the muscular coat. Three also showed ulcerations in the pharynx, but this is not infrequent. In 1850, Jenner[3] reported a series of seventeen autopsies with one case of ulceration. In 1891, Holscher[4] reported a series of two thousand typhoid autopsies without a single case of ulceration, in fact the only ones showing any esophageal involvement at all were one case of gangrene and two of necrosis of the laryngeal cartilages with perforation into the esophagus. In 1893, Ouskow[5] reported four hundred and thirty-nine autopsies with several cases of ulceration of the pharynx but none of the esophagus. Riesman[6] describes the case of a young man who succumbed to typhoid about the third week of the disease, (eight days after admission to the hospital) who, during his illness, presented no symptoms referable to the esophagus, but on autopsy, a perpendicular chain of ulcers, four in number, were found on the anterior wall of the upper end of the esophagus. The ulcers were well defined, the largest measuring 7x4 mm. Mitchell[7], in a series of fifty-six autopsies, discovered one case of ulceration of the esophagus and described it with minute detail. This case is also referred to by Osler[8] in a series of

*Presented as a Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Chicago, Ill., June 15 and 16, 1915.

eighty-three autopsies. This patient was a man, aged 28 years, who died on the eighteenth day of a very severe typhoid infection. Two days before death, he showed evidence of pain and difficulty in swallowing. On autopsy, the esophagus presented three well defined ulcerations on the posterior wall beginning about 3 cm. below the pharynx. The largest ulcer measured 1 cm. x 2½ mm., and two of the three extended down to the muscle. Microscopically, no typhoid bacilli could be found, "but histologically, the structure corresponds to what is seen in cicatrizing typhoid ulcers of the intestine." On the other hand, Berg[9], with a series of 1,626 typhoid autopsies, Studer[10] with 341 autopsies, Dopfer[11] with 927 autopsies, and Hoffman[12] with 250 autopsies, reported no lesions of the esophagus. Thus in a total of 5,729 autopsies, 11 cases of ulceration are mentioned.

Butler[13] reports the only case that gave definite symptoms of ulceration during the course of the typhoid and was followed by recovery. His patient was a young woman, aged 22 years, who developed a typical typhoid five weeks after a normal confinement. Early in convalescence, after an uneventful course, she developed "an uneasiness hardly amounting to pain" on swallowing. This however, soon increased to a definite pain, referred to a point just above and to the left of the episternal notch. At this time there was ulceration of the left tonsil and posterior pharyngeal wall. On the fourth day of the dysphagia, a swelling developed over the episternal notch and the inner end of the left clavicle, and in a few days the entire area bounded by the left ear, left axilla and episternal notch was swollen, somewhat reddened and edematous, and very painful on pressure or movement. At this time the dysphagia was very severe. The esophagus was given complete rest, the patient fed per rectum, and in a week the process subsided and she recovered. The diagnosis was made upon the similarity of the symptoms to those seen in cases of ulceration of the upper esophagus due to malignant growths.

As to the cause of such ulcerations, there has been considerable discussion. As yet no one has demonstrated that they are due directly to the typhoid bacilli, as are similar ulcers in the intestine. Mitchell,[7] in the report of his case, is the only one to call attention to any histological similarity. It has been claimed that such ulcerations are ordinary simple or peptic ulcers, such as are frequently noted in autopsies upon those having succumbed to slowly debilitating diseases, such as carcinoma, tuberculosis or chronic sepsis. Such an explanation might hold good for those occurring in the

lower part of the esophagus but could hardly be valid for those found in the upper end. In only two of the stricture cases reported are there lesions of the upper part of the esophagus, and in both of these cases, strictures were found at the lower end as well. It is at least quite probable that the formation of ulcers is favored both by the low-grade inflammation from which the whole of the digestive tract suffers, and by the local stasis of blood which is increased by gravity. That varicosities of the esophagus occur principally in the upper and lower parts of the tube is well known, and the formation of minute thrombi in such vessels may be a causative factor. The corrosive action of medication has been advanced as a cause of the ulcers, but there is no evidence to support this theory. On the other hand, it is not to be doubted that all lymphatic tissue is especially susceptible to typhoid, and the typhoid bacilli have been found in the lymphoid tissue of the larynx and pharynx. No case of a typhoid ulceration having perforated the esophagus has been found at autopsy.

There are no classical symptoms by which the condition may be diagnosed. The majority of the cases have first noticed difficulty in deglutition during the typhoid convalescence or even later. Dysphagia is mentioned by several observers as occurring during the course of the typhoid, but this symptom may also be due to ulceration in the pharynx or larynx. Pain in the epigastrium has been a prominent symptom in four of the stricture cases, but this occurs in many uncomplicated typhoids. Haematamesis was noted in two cases (Packard's[14] and Dugan's[21]) but this symptom is seen in many cases who on autopsy show no lesion of the esophagus.

Only seventeen cases of post-typhoid stricture are on record. Of these, two occurred in females (Osler's[8] and Roberts'[22]) the remainder being in males under the age of 36 years. The time of development of the difficulty in swallowing varied from the second to the twelfth week after the onset of the fever, but was most often noted about the fourth week. As was above remarked, only two of the cases showed any stricture formation in the upper part of the esophagus, and in both of these cases strictures were also found in the lower end. It is a singular fact that all of the cases have been reported by American observers, no European reports having been found after a most careful search.

To the list of reported cases the writer desires to add another, the patient a young man, aged 20 years, previous history negative. During February, 1914, he suffered with a typical attack of typhoid during which the diagnosis was confirmed by Widal and Diazo re-

actions. At the end of the fourth week he was allowed soft diet and noticed difficulty in swallowing soft toast. This difficulty increased and by the middle of April he could take no solids whatever, the food being regurgitated in a few minutes. In June, when he presented himself for esophagoscopy, he was able to take fluids slowly, and had lost considerable weight. The x-ray showed a tight stricture at the level of the eighth dorsal vertebra with a large dilitation above. (Fig. 1). The Wassermann and von Pirquet reactions were entirely negative. On direct examination, a tight stricture was found which was passed with a probe with considerable difficulty.

To exclude the possibility of the stricture being spasmodic, a general anesthetic was administered but the condition remained unchanged during this examination. Within three weeks the stricture was dilated sufficiently to permit the introduction of a Lerche dilator, after which it was dilated rapidly and easily to normal size. Four months later he was able to swallow any food, but occasionally he noticed a slight hesitation in its passing the site of the former stricture, undoubtedly due to the fact that the dilitation of the esophagus had not completely contracted and thus lacked the normal propelling power.

The following is a brief resume of the cases reported to date in the order of their publication:

Packard's Case.[14]. (Also quoted by Keen[15]). Male. Age, 35 years. Severe typhoid with hemorrhages from the stomach and

bowels, having vomited blood on three occasions. During the course of the typhoid, difficulty in swallowing capsules was noted, so this form of medication was discontinued and no further trouble of this kind was experienced until the tenth week, when the patient was allowed soft diet. During the twelfth week, an esophageal examination disclosed three strictures, the first 12 cm., the second 25 cm., and the third 31 cm., from the teeth. The lowermost stricture was only slightly dilated after a month's effort. Three months later he appeared at St. Vincents Hospital in New York with a stricture 9¾ inches from the teeth, through which the smallest bougie could not be passed, so a gastrostomy was performed.

Hughes' Case.[16] Sex ? Age ? Developed difficulty in swallowing three months after the onset of typhoid. At seven months, it "was impossible to swallow anything liquid or solid except very occasionally a small quantity of water." No pain or discomfort noted. The stricture could not be passed with bougies, so a gastrostomy was done and at the same time an unsuccessful attempt was made at retrograde dilitation. The patient died shortly after the operation. An incomplete post-mortem was made and a stricture found two inches above the pylorus extending upward for two inches "and probably as much more." The constriction was due to a fibrous and muscular overgrowth entirely outside the mucosa and the mucous membrane showed no evidence of scars.

Summers' Case.[17] Male. Age ? At the end of the second week of typhoid, the patient complained of severe epigastric pain and was unable to eat for two days, after which time he had difficulty in swallowing. The fever subsided at the tenth week, shortly after which the patient noticed that food did not always reach the stomach, sometimes being regurgitated. This increased rapidly so that in a few weeks it required from thirty to sixty minutes to swallow a teacupful of fluid. On examination a stricture was found 30 cm. from the teeth which admitted a filiform but after a month of unsuccessful effort at dilitation, Abbe's operation was performed with complete success.

Pyle's Case.[18] Male. Age ? Typhoid lasting eleven weeks. During the course of the fever (the exact time not stated) the patient developed pain and difficulty on swallowing, which was intensified by acids or acid foods. The pain gradually subsided, but the difficulty in deglutition increased to a point where fluids would pass only with difficulty. Examination disclosed a stricture at the level of the esophageal opening of the diaphragm. A bougie

1/16 in. in diameter was passed and the stricture was dilated by an original method, using rubber bags inflated with water.

Tinker's Case.[19] Male. Age 31 years. Severe typhoid lasting five months. First difficulty in swallowing noticed in the eighth week, but it progressed very slowly. At the end of a year, patient was having difficulty even with liquids, sometimes as long as four days passing without fluids of any kind being taken, but after such periods the stricture would relax and fluids again pass. A stricture 29 cm. from the teeth was located. Before this examination was made, a gastrostomy had been done in another city and shortly after the patient disappeared.

Osler's Case.[20] Female. Age 35 years. Typhoid fever and relapse, duration seven weeks. At the third week, had an attack of gastritis lasting ten days, during which there was considerable vomiting but no signs of blood in the vomitus. On first attempting to swallow fluids, she complained of a severe pain referred to a point below the outer third of the right clavicle, but this was relieved by the passage of a stomach tube, after which the pain was referred to the right costal border. The difficulty in swallowing increased, so that by the time she presented herself for examination, eighteen months after the typhoid, she was able to take only fluids, all solids regurgitating within five minutes. A stricture was found 34 cm. from the teeth allowing only the smallest bougie to pass, but was dilated to 23 F. within a month.

Dugan's Cases.[21] These were in a family of whom four developed typhoid and two developed strictures.

Case 1: Male. Age 18 years. Severe typhoid of eight weeks' duration, during which he had severe pain in the chest and stomach but showed no signs of difficulty in swallowing. By the twelfth week a stricture had so rapidly formed that the smallest bougie could be passed only fourteen inches and the patient died eighty-five days after the onset of the typhoid.

Case 2: Was in an older brother, aged 22 years. Typhoid with relapse, fourteen weeks' duration. During the relapse he had several hemorrhages from the bowels and vomited blood. The exact time of the development of the stricture is not stated, but when first seen for this condition he had been unable to swallow liquids for four days. His weight had been reduced from 150 to 85 lbs. Gastrostomy was immediately done after which he was again able to swallow fluids.

Roberts' Cases.[22] *Case 1:* Young male.

Case 2: Young female. No definite data given in either of these cases except that it was impossible to pass the smallest bougies, in both operation was advised and refused, and both patients died.

Case 3: Male. Age 15 years. Typhoid of nine weeks' duration. Toward the end of his illness he choked on attempting to swallow a capsule, and within ten days he was unable to swallow even liquids. A stricture was found twelve inches from the teeth which was gradually dilated with bougies.

Price's Case.[23] (Quoted by Dr. McMurtry). Male. Age ? Severe typhoid with frequent hemorrhages. Duration about one hundred days. A stricture developed eleven inches from the teeth, and a gastrostomy was done. At the time of operation, the stricture was dilated from below, followed by bougies from above.

Wilson's Case.[24] No details of the case reported, it simply being stated that the patient developed a stricture following typhoid, and that it was dilated with bougies in about four weeks.

Turner's Case.[25] Male. Age 16 years. Severe typhoid with delirium. During the fourth week he complained of difficulty in swallowing capsules and three weeks later in taking solid food, which was regurgitated with slight hemorrhage. An obstruction was found ten inches from the teeth. Six months after the onset of the fever he was very much emaciated and when seen by Turner had been unable to swallow anything for about ten days, having been fed per rectum during that time. A small bougie was passed and at the lower end of the esophagus two strictures were located about half an inch apart. These were successfully dilated with bougies.

(Two other cases of stricture following typhoid are reported by Dr. Turner, but both were undoubtedly due to spasm and both recovered spontaneously).

Thompson's Cases.[26] *Case 1:* Male. Age 17 years. Severe typhoid during the fourth week of which patient complained of difficulty in swallowing and in a few weeks only liquids could be taken. Nine months later the stricture had so contracted that it required a half hour for a glass of water to pass. The stricture was found to be just above the cardiac orifice and within fourteen weeks it was dilated to a diameter of 14 mm. He then developed pain and soreness at the site of the stricture, fever, cough, and the difficulty again increased. He died about three months later.

Case 2: Male. Age 26 years. During the fourth week of typhoid, he developed difficulty in swallowing capsules. At ten months, he had developed a very tight stricture, having taken no fluids for three days when first seen by the writer. On examina-

tion, a stricture was located 15¼ inches from the teeth, a 4 mm. bougie was passed and the stricture gradually dilated.

Case 3: Male. Age 26 years. Severe typhoid of seventy-two days' duration. First difficulty in swallowing noticed when capsules were taken about the end of the first month. At the end of a hundred days, no solids whatever passed into the stomach, being regurgitated sometimes several hours after ingestion. On examination, four and a half months after the onset of the typhoid, one stricture was located 8 inches and another stricture 13½ inches from the teeth. Both were dilated with bougies.

Thus, in the above series, gastrostomy was performed upon six cases with one death, eight were dilated with bougies with one death, and three died apparently without either operation or dilitation. These cases were practically all before the perfection of esophagoscopy, but at the present time the mortality should be practically nil, and if the patients are seen before severe starvation develops, there will be no indication for a gastrostomy.

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247 Madison Street.

REPORT OF A CASE OF AN EXCESSIVE BLEEDER.

Dr. Lee M. Hurd, New York.

Mr. G. Age, 27. Lawyer. A thick set, powerfully built man, who engaged in athletics while at college. Weight at college, 160 pounds. Now his weight is 180 pounds. He is five feet, seven inches in height.

Family history reveals no person that bleeds excessively. He has had measles and pneumonia, epistaxis since about his fifth year, lasting several hours every three to six months, sometimes from blowing nose, sometimes spontaneously; very difficult to check, at which times he usually used some astringent or adrenalin.

Tonsillotomy at 6 years of age with no alarming hemorrhage. When 12 years of age, he had four six-year molars extracted at 11 a. m., bled all day and that night, in sleep, stomach would fill with blood. Dr. Russell Bellamy plugged tooth sockets which stopped bleeding, gave saline solution, intravenously. The patient was very weak afterwards.

When 15 years old, while skee-running, he fell, landing upon a pair of nippers he had in his hip pocket. A large hematoma formed, which was opened, and about one pound of blood clot was removed.

Three years later, while playing foot-ball he ruptured another vessel in his left leg, which was very painful, and the hematoma was absorbed. Hand and head cut while playing foot-ball, both bleeding normally and ceasing spontaneously.

I saw Mr. G. first on April 28, 1914, complaining of a thick post-nasal discharge, recurrent tonsillitis, and one tonsil much swollen. Nasal septum deviated to left with slight erosions that could account for the epistaxis, large tonsillar stumps.

I had Dr. L. W. Strong examine his blood for clotting time, which he found to be normal, 4 minutes by capillary pipette method.

May 20, 1914, under hyoscine, morphine, and alypin anesthesia, I did a submucous resection of the septum with but slight flow of blood. The nose was packed, and the packing was removed at the end of 24 hours without hemorrhage.

The report of the coagulation time with this result seemed to assure me that the tonsils could be removed without danger of excessive hemorrhage.

May 29, 1914, under morphine, gr. one-fourth, atropin, one one-hundred-fiftieth, and ether anesthesia, the tonsils were removed by dissection and snare. Capsule very fibrous, moderate primary hemorrhage, and to guard against secondary hemorrhage, I tied off four bleeding points in each fossa. The patient left the operating table at 2:30 p. m. with a dry throat. At 3:00 p. m. vomited blood, 4:30 vomited blood. Given morphine, gr. one-fourth. 5:00 p. m. still vomiting blood. 6:00 p. m., pulse 120. 7:00 p. m., pulse 130. Started Murphy Drip; 8:30 drip discontinued. Pulse weak. Applied pressure with gauze sponges on forceps to both fossae; 10:00 p. m., vomited large amount of blood. Given morphine gr. one-fourth, normal horse serum, 10cc. 10:30 p. m., coughed out large clots. 11 p. m., vomited blood; 12, vomited again, pulse weak.

April 30, 8 a. m., hemorrhage had ceased. During evening, began to ooze again. Used pressure with sponges to fossae. House officer had been using tannic acid. At midnight, unable to count pulse. Temperature, 103.4. Used Murphy Drip. Sent for sister, prepared to transfuse. 4 a. m., restless. Morphine gr. one-fifth. Sister arrived, but hemorrhage had ceased. Temperature 102, pulse 108, resp. 16.

April 30, one dose of coagulose.

May 1, no hemorrhage. One dose of coagulose.

May 2, hemorrhage from left tonsillar fossae. Coagulose applied to fossae and two doses given. 9 p. m., vomited large amount of blood. Given, morphine, gr. one-sixth. Three Michel metal clips applied to left side.

May 3, coagulose given. Vomited milk with some fresh blood.

May 4, coagulose given.

May 5, coagulose given. 7 p. m., slight bleeding, right side. Considerable bleeding during the day. Pulse 130. Temperature 101.5, resp. 20.

May 6, coagulose given. 7 a. m., bleeding, house officer applied ice to neck, morphine gr. one-fourth. Cat gut ligatures applied at the time of the operation gave way, hemorrhage from both fossae followed, two more metal clips on the left side and three applied on the right side. Considerable bleeding during the day. Pulse 130, temperature 101.5, resp. 20.

June 10, two clips removed, left side.

June 20, one clip removed. Patient bleeds from the pin pricks of the clips.

July 3, remaining clips removed.

January, 1915, applied fused silver nitrate to the pocket in the pharyngeal vault. The crust resulting, the patient wiped off with a bent cotton applicator, which produced a slight hemorrhage, lasting three days.

The case clearly shows that there is another condition that causes excessive bleeding than the demonstrable changes in the blood that make up the picture of hemophilia.

This man's blood promptly clotted not only in the laboratory tests, but also in his throat, and even after it had reached his stomach. The condition seems to be that the intima of his vessels and the cut surfaces would not allow the clotted blood to adhere.

Also that coagulose had no noticeable effect.

Feb. 28, after using a post-nasal cotton applicator, which he is in the habit of doing, blood began to flow from the vault of the nasopharynx. I had him in the office from 9 a. m. to 1 p. m., finally staying the hemorrhage with the application of Thrombokinase. In about two hours, the hemorrhage started afresh, but the patient did not notify me until the following morning, when he was again exsanguinated. I then put in a post-nasal plug, removing him to the hospital. The plug was removed 24 hours later.

15 East Forty-eighth Street.

Through the courtesy of Prof. W. H. Howell of Johns Hopkins University, the patient's blood was examined.

The letters of Prof. Howell are appended with his permission.

COPY.

My dear Dr. Hurd:

Mr. G.'s blood has been examined in my laboratory on two occasions. The first examination was made by Dr. Denny, the second by myself. The points determined were the coagulation time (blood taken by vein puncture), the prothrombin time (the clotting time of oxalated plasma upon recalcification), the relative amount of antithrombin, and the power of tissue-extract (Kephalin) to activate the first serum.

In the first examination, the clotting time was prolonged beyond the normal for the method used. In the second examination, made 6 weeks later, the coagulation time was normal.

In both examinations, the prothrombin time was considerably prolonged and the antithrombin was somewhat above the high normal limit.

In the first examination, the Kephalin solution failed to activate the first serum, but in the second a normal reaction in this respect was found. I have some doubts about the first examination as regards this factor, since unfortunately the reaction of the testing reagents was not verified simultaneously upon a control serum; on the other hand it may be possible that Mr. G.'s blood may vary from time to time; only repeated examinations could settle this point.

The two examinations therefore give no positive indication except the presence of what might be called a moderate hemophilic tendency, as indicated by the low amount of prothrombin and the somewhat high amount of antithrombin in the plasma.

I hope it may be possible to repeat the observations at certain intervals to ascertain whether these factors vary.

Very truly yours,

W. H. HOWELL.

Lymphoid Growths of the Laryngopharynx. W. E. CASSELBERRY, *Jour. Amer. Med. Assn.*, Feb. 13, 1915.

The lymphoid follicles on the rim of the larynx undergo hyperplasia and develop into growths which occupy the laryngopharynx. Heretofore they have been of uncertain origin and were described as "lymphoma." In two cases the author has found definitely by cultural, microscopic and vaccine methods that these are of microbic origin. He has also found the infiltrating type of hyperplasia at the Eustachian orifice to be of the same nature. In these cases leukemia was excluded by the absence of glandular involvement and malignancy was excluded by microscopic examination, which showed, in each instance, simple hyperplasia of lymphoid tissue.

The favorite sites for this type of hyperplasia are (1) the vicinity of the Eustachian tube; (2) the lateral angles of the pharynx; (3) the tonsillar region; (4) the tonsil itself; (5) the larynx.

ED.

A DIRECT LARYNGOSCOPIC SPATULUM.*

DR. OTTO GLOGAU, New York.

Ever since the vocal teacher Garcia first demonstrated a way of examining the interior of the larynx, the medical profession tried to improve his method. For diagnostic purposes the study of the inverted picture of the larynx as reflected in the laryngeal mirror proved to be a wonderful achievement. The performance of intralaryngeal operations was, however, quite difficult in many instances. The goal to be reached was the replacement of this indirect method by the direct inspection of the larynx and deeper respiratory passages. Kirstein first applied the so-called autoscopic spatulum by means of which the epiglottis and the base of the tongue were forcibly pushed backward to such a degree that the laryngo-tracheal tube could be directly inspected through the mouth. There was, however, no provision made for retaining the spatulum in position. It was, therefore, practically of no value in the performance of intralaryngeal operations. Killian first exposed the interior of the larynx and of the bronchi by introducing electrically illuminated metal tubes. The lumen of the metal tube emerging from above the upper teeth represented the straight continuation of the laryngo-tracheal tube. This ingenious procedure was later improved by Jackson, Bruenings and Kahler, and is still very much in vogue. The method of direct inspection of the larynx was revolutionized by Killian's so-called suspension laryngoscopy. While in the other procedures the epiglottis and the base of the tongue were forcibly pushed backward, they were in Killian's new procedure automatically suspended on a spatulum that was attached to a kind of gallow arrangement. The interior of the larynx is perfectly exposed and directly accessible to operative interference. The suspension apparatus of Killian is, however, very expensive and rather complicated. Its use is almost exclusively limited to hospital work.

During my last stay in Berlin I saw the laryngeal spatulum of Struycken. The underlying idea of this instrument was the pushing backward of the epiglottis and the base of the tongue by lowering the spatulum on a fulcrum in the middle of the mouth. The construction of the instrument was, however, such a primitive one that the spatulum always slipped from its fulcrum whenever the

*Demonstrated before the New York Academy of Medicine, Section on Laryngology, January 27, 1915, and before the American Academy of Otolaryngology, Chicago, October 6th, 1915.

instrument was introduced. Besides, the size and shape of the spatulum were such that an exposure of the larynx could not be obtained. The principle of moving the spatulum on a fulcrum in the center of the mouth struck me as a good one, and on this principle I constructed a handy and inexpensive instrument for the direct inspection of the larynx that combines the advantages of both Kirstein's autoscopic spatulum and Killian's suspension laryngoscope.

The latest model of the instrument consists mainly of the palate plate (D), the tongue depressor, consisting of the tongue blade (B) and the handle (A), and of the spatulum (C), gliding within a sliding arrangement (H) of the tongue blade. The sliding spatulum may be fastened in any position by means of the screw (L). At

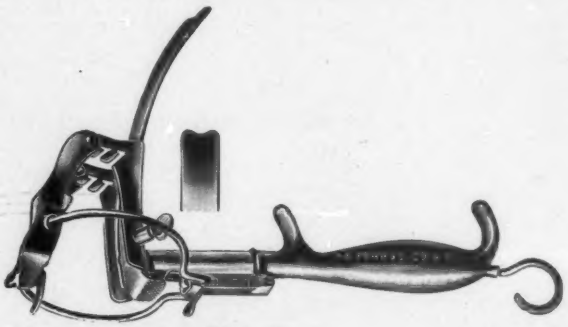


Figure 1.

the innermost end of the tongue plate there is attached under a right angle an arrangement of teeth (G and G^1), into which the connecting knobs (F and F^1) of the palate plate fit. The joint thus formed represents the fulcrum within the center of the mouth upon which the spatulum turns. At the outside end of the palate plate (D) there are two small universal joints in which a heavy metal ring is movable. The narrow center-piece of this metal ring fits into the teeth arrangement (I) on the handle (A). By closing the lever (K) and fastening it through the spring (M), the palate plate becomes immovably attached to the tongue blade and the handle. The instrument may be introduced under local or general anesthesia. In the upright position of the patient, the head is bent backward as far as possible. According to the size of the mouth, the knobs (F and F^1) of the palate plate (D) are locked into the lower or upper teeth of the fulcrum (G and G^1). The metal ring (E) is then connected with the lowest notch of the teeth arrange-

ment (I) and fastened in this position by means of the lever (K) and the spring (M). The operator stands in front of the patient. The patient is told to keep the tongue pulled forward. The instrument, after being closed in the above described way, is then introduced till the palate plate rests on the hard palate in such a way that the upper teeth are just inside of the vertical teeth shield that forms the outer bounding line of the palate plate. The spatulum (C) is then introduced into the sliding arrangement (H) of the tongue blade (B) and made to glide downward alongside of the back of the tongue and over the epiglottis. The spring (M) is then opened, the lever (K) released and the metal ring (E) moved upwards as far as possible and then fastened to the uppermost notch possible of the teeth arrangement (I), by closing again the lever (K) on the spring (M). Due to the curvature of the tongue blade and of the spatulum, the notch-shaped inner end of the latter is

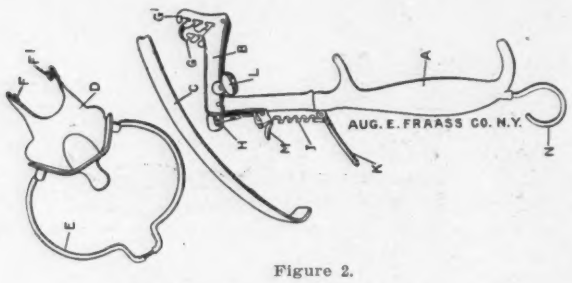


Figure 2.

about two inches below the level of its outer end. Thus the tongue and the epiglottis are pushed backward without any force by the construction of the gliding surface alone.

In the recumbent position of the patient, the head hangs over the table and is bent backward and downward. The operator stands behind the patient. The instrument is introduced in a way similar to the above described. Due to the position of the patient, the tongue-blade will then form the roof of the exposed cavity and the palate plate its floor. By introducing the sliding spatulum, the tongue and the epiglottis will become suspended on the end of the spatulum. Both in the upright and recumbent position a full exposure of the interior of the larynx and the vocal cords is obtained. The instrument, though self-retaining, may best be held in position by means of the handle (A). In order to expose the anterior commissure, the handle must be pushed further upward in the upright

position of the patient and further backward in the recumbent position. In general anesthesia, the tongue must be pulled forward during the introduction of the sliding spatulum.

Figure 3 shows the exposure of the interior of the larynx in the recumbent position of the patient. The wide access to the exposed parts permits of easy operative interference. Straight instruments are used. Under guidance of the direct laryngoscopic spatulum, the

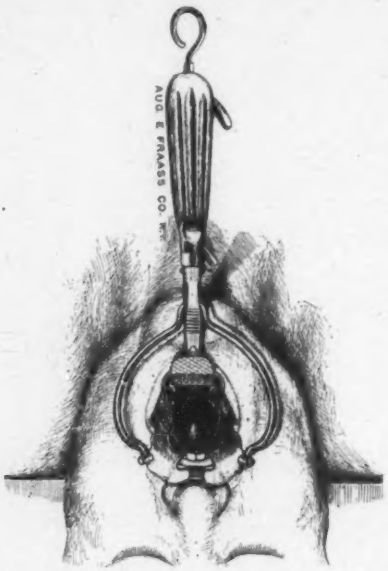


Figure 3.

writer curetted the posterior laryngeal wall in two cases of laryngeal tuberculosis and removed in three cases papillomata from the vocal cords.

The illumination, though sufficient with the ordinary head-mirror and reflected light, is best obtained by an electric head-light, such as Kirstein's lamp, etc., or by attaching a small electric bulb directly to the palate plate. The instrument is made by Aug. E. Fraass Co., N. Y.

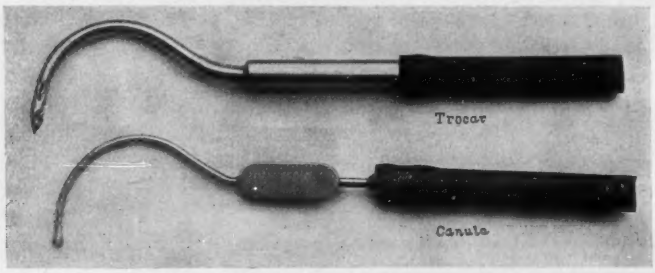
1320 Madison Avenue.

ANTRUM TROCAR AND IRRIGATING CANULA.*

DR. GORDON B. NEW, Rochester, Minn.

The two instruments herein described were suggested because of the difficulty first in securing satisfactory instruments for tapping antra through the lateral nasal wall under the inferior turbinate to drain pus and for diagnostic purposes, and second, a suitable canula which could readily be passed into the same opening for washing out the antrum.

The trocar is a sharp, pointed, curved tube with but one opening running along its entire length and with no channel for return flow. There are two openings, one on either side of the point of the trocar. A heavier portion is at the opposite end to give more strength and a handle to the instrument. To this is attached a piece of rubber tubing. The trocar is of sufficient strength so that it will not bend with



the necessary amount of force employed. Its curve is such that if the tip of the instrument is placed under the inferior turbinate anteriorly, the handle rotated towards the mid-line and the force employed directed upward, outward and backward, the trocar will readily pass into the antrum at the thinnest portion of the lateral wall, about half way back in the nasal passage. A syringe with catheter tip may be attached to the rubber tubing and the antrum irrigated. If it is necessary to irrigate the antrum, in a few days the irrigating canula, a Killian canula bent in the same shape as the trocar, is most useful, since it is a little smaller in diameter and it readily passes into the same opening without the necessity of feeling along the lateral nasal wall to locate it. By this method no local anesthetic is required. These instruments have been used at the Mayo Clinic for the last three years with entire satisfaction.

*From the Mayo Clinic.

BOOK REVIEWS.

Nursing in Diseases of the Eye, Ear, Nose and Throat. By the Committee on Nurses of the Manhattan Eye, Ear and Throat Hospital, New York City. Second edition, thoroughly revised; 12 mo. volume of 291 pages, illustrated. W. B. Saunders Company, Philadelphia and London, 1915. Cloth, $1.50 net.

There is a likelihood that in the near future the profession of nursing will undergo specialization in the same manner as the practice of medicine has done so and from the very nature of the case we would expect a nurse devoting her time to pediatric nursing or to oto-laryngological nursing to be more competent in understanding the peculiar problems of her specialty than one doing general work. There has always been a demand for a book on nursing devoted to diseases of the eye, ear, nose and throat and the book under consideration is certainly the most up-to-date in its field. It is written in a very lucid style and no nurse, in view of the training she must go through, should have difficulty in digesting its contents thoroughly. A glance at its contents will show its scope. Its chapters take up in order (1. General part) the germ theory of disease, antiseptics, disinfection of sick-room and clothing, sterilization of hands, dressings and instruments, preparation of surgical dressings for nose and throat work, preparation of operating room, the nurses' duties at operations, the nurses' duties in emergencies, management of troublesome children, the feeding and care of infants. Part II takes up the anatomy and physiology of the eye, common remedies used in the treatment of the eye (antiseptics, astringents, anesthetics, mydriatics and myotics, etc.), eversion of lids, contagious eye diseases, eye instruments. Part III outlines the anatomy and physiology of the ear, general methods and instruments commonly employed in the examination of the ear, tests for hearing and functional tests of the static labyrinth, diseases of the auricle and external auditory canal, diseases of the middle ear, preparation for the mastoid and other operation, diseases of the internal ear. Part IV considers the diseases of the nose and accessory sinuses, and Part V the pharynx and larynx (methods of examination, tumors, foreign bodies, tuberculosis, douches, sprays, gargles, tracheotomy, etc.). The book is very well illustrated and is to be highly recommended. ED.

Einführung in die Musikwissenschaft auf Physikalischer, Physiologischer und Psychologischer Grundlage. von Karl L. Schaefer. Druck und Verlag von Breitkopf und Härtel, Leipzig, 1915. M. 4, in Leinenband, M. 5. Pp. 165.

The book opens with the question, "Was ist ein Ton?" and goes on to say that "die Antwort auf diese fundamentale Frage der physikalischen Akustik lautet: Es ist der Effekt pendelartiger Schwingungen (Oscillationen, Vibrationen), die kleinsten Teilchen irgend eines festen, flüssigen oder gasförmigen Körpers um den Punkt ihrer Ruhelage im Gleischgewichtszustande ausführen." There then follows a detailed consideration of the questions of amplitude, molecular power and elasticity, longitudinal, transversal and torsion tones; in short, a very complete study of the physics of sound. Tone-color and the principles of all the musical instruments are analyzed. The book should prove valuable for the student of music and principles of orchestration. It contains a fairly comprehensive chapter on the voice, its production and qualities, and another on the anatomy and physiology of the ear and the theories of sound perception. As its title denotes "Introduction to the Science of Music," it is rather technical in its scope, and for the student of the science of music rather than the art of performance it contains much that is of value. ED.

MEDICAL NEWS.

The Nobel prize for medicine for 1914, of the value of about $40,000, has been awarded to Dr. Robert Barany, privat-docent in otology in the University of Vienna, for his work on the physiology and pathology of the vestibule of the ear. The prize for medicine for 1915 is reserved until next year.

Since the fall of Przemysl, Dr. Barany has been a prisoner of war at Merw, in the Trans-Caspian district of Russia.

Early Diagnosis of Cancer of the Esophagus. WILLY MEYER, *Amer. Jour. Surg.* V. 29, No. 7, July, 1915.

Meyer describes the technique of operation for removal of a cancer of the esophagus. The operation consists of three stages: (1) Gastrostomy and careful determination of the mobility of the tumor around the cardia, the extent of glandular infiltration, etc. The Beck-Jianu operation is preferable when the esophageal growth is situated behind or above the aortic arch and there are no infiltrated glands at the cardia. This operation is preferable because it combines inferior partial esophagoplasty with gastrostomy. The Beck-Jiann operation cannot be performed if the cancer is located in the lower portion of the thoracic esophagus, or it involves the hiatus esophagus or the cardia itself. (2) Thoracotomy; division of the esophagus above the tumor within healthy tissue; extraction and transposition of the proximal stump downward through an esophagotomy wound made at the neck. (3) Removal of the tumor from below after the stomach has been divided transversely and its distal portion closed, the remaining pouch being in communication with the gastric fistula.

So far as early diagnosis is concerned, Meyer emphasizes the fact that from 80 to 90 per cent of cases in which there is gradual, steadily increasing difficulty in deglutition, are carcinomatous, and every case of esophageal stricture where benign stenosis due to the swallowing of a caustic can be ruled out at once, should be considered as highly suspicious of carcinoma. ED.

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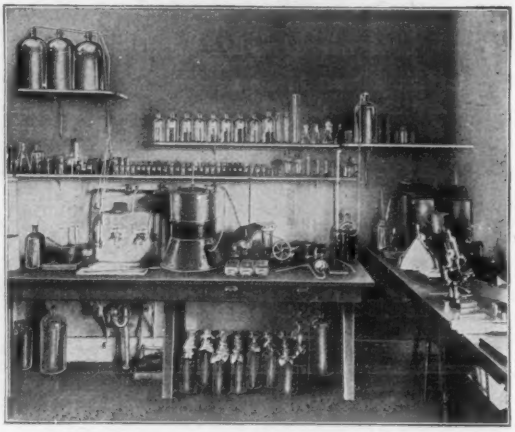

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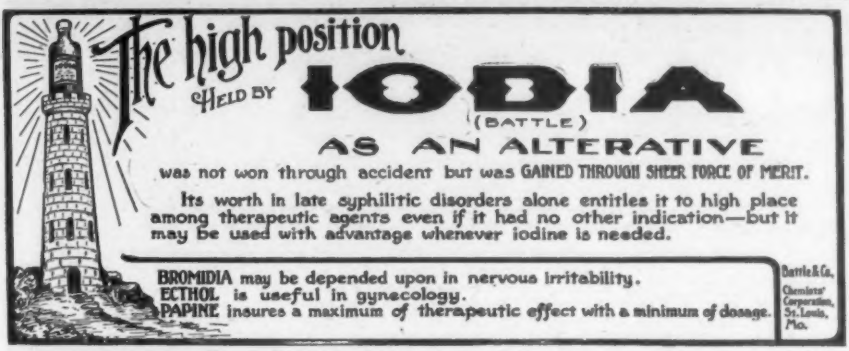
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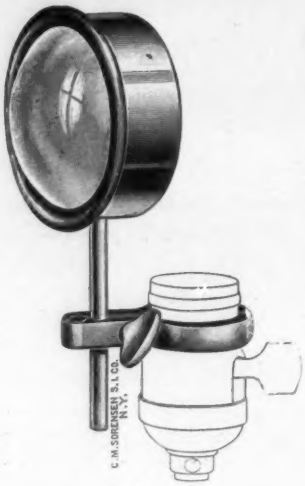

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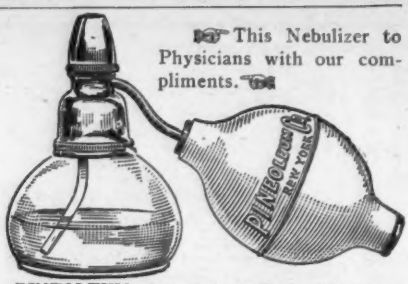

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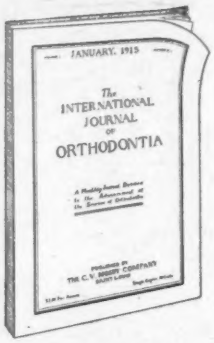

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